VOLUME 14 NUMBER 5

AMERICAN JOURNAL

DISEASES OF CHILDREN

EDITORIAL BOARD

NOVEMBER, 1917

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THE CONDITION OF THE REFLEXES IN POLIOMYELITIS *

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The most constant symptom of poliomyelitis — and the one which should always lead us to think of the possibility of that disease, especially during epidemics — is an alteration in the reflexes accompanying or following a short febrile period.

Various changes in the normal reflex response have been described by numerous investigators, more especially by Wickman, Muller, Zappert, Foerster, Neurath, and recently by Draper, Peabody and Dochez.[1] In studying the large number of cases admitted during the epidemic of 1916 to the Kingston Avenue Hospital, we have encountered some interesting facts to which we shall take the liberty of drawing attention.

* Submitted for publication Sept. 8, 1917.
* From the Kingston Avenue Contagious Disease Hospital.
* The author wishes to express his appreciation for the cooperation of Dr. Robert J. Wilson, Director of the Bureau of Hospitals, Department of Health, New York; the members of the resident, and intern staff of the Kingston Avenue Hospital, and also to the following members of the visiting staff: Drs. L. C. Ager, E. S. Dalton, J. P. Pendleton, S. Feldstein, M. B. Gordon, P. W. T. Moxom, H. L. Ratnoff, J. M. Wallfield and J. F. Crawford.

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The Condition of the Patellar Reflex.—The knee jerks are the most frequently affected of all reflexes. This is no doubt to be attributed to the common paralytic involvement of the quadriceps extensor group of muscles. In order to show the frequency of alterations in this reflex Table 1 should be instructive, as it has been based on 818 cases of poliomyelitis, most of the patients being admitted in the early paralytic stage. If summarized it will be found that the knee jerks were altered in some way in 81 per cent. of the 818 cases classified.

TABLE 1.—THE CONDITION OF THE PATELLAR REFLEX IN 818 CASES OF POLIOMYELITIS

	Right Knee Jerk	Left Knee Jerk
Absent in	549	538
Diminished in	68	67
Exaggerated in	38	34
Sluggish	9	17
Altered in	664	656
Normal in	154	162
Total cases	818	818

Exaggeration of the Knee Jerks.—Ed. Muller and Wickman[2] have drawn attention to the initial increase, which may precede the onset of paralysis. We have met this change in most all the cases seen early in the preparalytic stage, and we strongly believe it is the usual, if not invariable, change found in the first twenty-four hours of the meningeal phase of the disease. Exaggeration would naturally be expected to precede the later diminution and absence, since it is recognized that the first stage of inflammation is accompanied by increased functional activity, and that this principle applies to the tissues of the nervous system as much as it does to those of any other part of the body. Another factor concerned possibly is that the normal inhibitory influence of the brain through the upper motor neuron is no doubt temporarily diminished by the inflammatory changes which are often present to a certain extent throughout the entire cord.

The reaction is often so marked that the slightest tap with the percussion hammer will elicit a response which will move the foot through an arc of from 30 to 60 degrees. As the preparalytic stage advances this exaggerated response becomes diminished and finally

2. Wickman, Ivan: Nervous and Mental Disease Monograph, Series, No. 16, New York, 1913, p. 48.

disappears when paralysis becomes definitely manifest, and for this reason it often passes unnoticed unless the patient is seen early.

Hyperactivity may, however, persist into the stage of paralysis in a certain proportion of the patients, and most often in those in whom the paralytic involvement is slight. To show the limb or limbs affected and the extent of the paralysis under such circumstances, thirty cases with exaggerated patellar reflexes at this period of the disease have been tabulated in Table 2. When this table is examined two important facts are noticable: (1) the relative infrequence of any marked paralysis in the entire series, and (2) the high proportion of the spastic form of paralysis present. In nearly all the thirty cases classified where the lower limbs were involved the paralysis was slight, for if it had been severe the quadriceps tendon would undoubtedly be affected

TABLE 2.—DEGREE AND TYPE OF PARALYSIS

Extremity Paralyzed	Total Number of Cases with Affected Extremities	Degree and Type of Paralysis			
		Weak	Partial	Complete	Spastic
Left upper	4	3	..	1	
Both uppers	9	4	2	..	3
Right lower	4	3	1		
Left lower	3	..	1	1	1
Both lowers	15	7	2	..	6
Totals	35	17	6	2	10

to such an extent that an absence of the knee jerk would be the result. Of the thirteen cases in which the upper extremities were concerned, in eight the paralysis was limited to that part of the body, the lower extremities being perfectly normal except for the exaggerated knee jerks. Such cases are extremely interesting and have been previously noted by Wickman,[2] and by Neurath, Zappert, Foerster and Ed. Muller. all of whom are cited by Wickman. Wickman concludes that they are due to a slight injury to the pyramidal tract in its course through the upper regions of the cord.

The large number — ten out of thirty-five — of spastic cases in the series indicates that the meningitic types of poliomyelitis are most apt to be accompanied by hyperactivity of the knee jerks, for spasticity usually belongs to that particular form of the disease.

In three of the thirty cases the knee jerks were entirely absent on the paralyzed side and exaggerated on the opposite or normal side. Wickman, Zappert, Draper and Peabody and Dochez have made an

identical observation in some of their patients. The pathologic basis for this symptom would seem to be that the inflammatory reaction around the cells of the anterior horns on the side with paralysis was sufficiently intense entirely to obliterate the reflex arc, while on the opposite side it was so slight as only to be productive of increased functional activity.

The relative frequence of hyperactivity varied in the different forms of poliomyelitis, as is shown in Table 4, giving, as it does, in percentage ratio, the occurrence of exaggerated, diminished, sluggish and absent reactions. It would tend to prove that an increased response was present most frequently in the meningitic and ataxic cases, and in the

TABLE 3.—SHOWING THE RELATION BETWEEN THE NORMAL AND ALTERED REACTIONS—

Type of Poliomyelitis	Right Knee Jerk			
	Normal		Altered	
	Number of Reactions	Relative per Cent.	Number of Reactions	Relative per Cent.
Single Types:				
Myelitic	72	21	267	78
Meningitic	3	23	10	77
Ataxic	20	41	29	59
Bulbar	9	50	9	50
Combined Types:				
Myelitic and meningitic	14	11	111	89
Myelitic and meningitic with hydrocephalus	0	..	13	100
Myelitic, meningitic and bulbar	3	18	14	82
Bulbar and meningitic	0	..	7	100
Bulbar and myelitic	1	5	19	95
Bulbar and myelitic with hydrocephalus	0	..	7	100

combined types of the disease in which meningitic symptoms were present, while it was unusual in the bulbar, bulbar myelitic, and myelitic cases. This last fact differs somewhat from the observations of Wickman and others, who noted that the knee jerks may be exaggerated if the bulb is involved. We think this difference lies in the fact that in the nine cases of our series in which there was an absence of the reflex, the children were mostly in rather poor condition, almost moribund, when examined, and we have only rarely been able to elicit a reaction under such circumstances. In the other nine cases, however, the response was normal, but no doubt had the examination been made in the first twenty-four to forty-eight hours of the disease an exaggeration would probably have been found.

We believe that while the bulbar forms of poliomyelitis may show hyperactivity of the knee jerks in the early meningitic phase, this soon changes to a more or less normal reaction, and later, in the terminal phase, disappears entirely.

Medin[3] noted exaggeration of the patellar reflex in many of the ataxic forms of the disease, but Wickman (p. 75) found a diminished or lost reaction in similar cases. We should be inclined to think with Medin that exaggeration occurs frequently, for we noted this change in 34 per cent. of the total alterations in the ataxic forms of the disease. Wickman (p. 48) has observed cases in which the reflex may be exaggerated in an atrophied and obviously paralyzed leg. Among our

—OF THE PATELLAR AND PLANTAR REFLEXES IN THE VARIOUS FORMS OF POLIOMYELITIS

Left Knee Jerk				Right Plantar				Left Plantar			
Normal		Altered		Normal		Altered		Normal		Altered	
Number of Reactions	Relative per Cent.	Number of Reactions	Relative per Cent.	Number of Reactions	Relative per Cent.	Number of Reactions	Relative per Cent.	Number of Reactions	Relative per Cent.	Number of Reactions	Relative per Cent.
65	19	272	81	144	56	110	43	146	58	106	42
3	23	10	76	5	55	4	44	5	55	4	44
24	45	30	55	33	76	10	23	30	76	9	23
8	44	10	55	11	64	6	35	11	61	7	39
18	14	107	86	56	54	47	45	54	56	42	44
0	..	14	100	5	45	6	54	7	64	4	35
3	17	14	82	5	35	9	64	5	38	8	61
0	..	8	100	6	85	1	14	6	85	1	14
1	4	20	95	10	62	6	37	8	61	5	38
0	..	7	100	1	16	5	83	1	16	5	83

cases we can recall three or four examples of a similar character, and in all the response was markedly increased.

Not infrequently in convalescence the knee jerks were found hyperactive when every other symptom of the disease had disappeared, the patient showing no paralysis of any description and walking in an apparently normal manner. This was probably indicative of the stage of improvement and had succeeded on a diminished or absent reaction, and just preceded the return to normal.

Diminution of the Patellar Reflex.—This often follows the exaggeration of the preparalytic stage, and just precedes the disappearance

3. Medin, O.: Cited by Wickman (Footnote 2), p. 75.

of the reflex with the onset of paralysis. Where the paralytic involvement is slight a diminished response is not infrequent throughout the entire course of the acute stage of the disease. During the stage of improvement it is usually the first change noticed. At times it just precedes the exaggeration which occurs before the reflex becomes normal, but more frequently the change occurs from the diminished to the normal response.

The relative occurrence of a diminished reaction in the paralytic stage varies in the different types of the disease, as is shown in Table 4.

TABLE 4.—SHOWING THE PATELLAR AND PLANTAR—

Type of Poliomyelitis	Right Knee Jerk				
	Normal	Exaggerated	Diminished	Sluggish	Absent
Single Types:					
Myelitic	72	11	32	4	220
Alterations expressed in per cent.*	..	4	11	1.5	82
Meningitic	3	2	2	1	5
Alterations expressed in per cent.	..	20	20	10	50
Ataxic	20	10	5	..	14
Alterations expressed in per cent.	..	34	17	..	48
Bulbar	9	..	..	..	9
Alterations expressed in per cent.	..	..	..	..	100
Combined Types:					
Myelitic and meningitic	14	12	6	3	90
Alterations expressed in per cent.	..	10.5	5.5	2	82
Myelitic and meningitic with hydrocephalus	..	2	1	..	10
Alterations expressed in per cent.	..	15	7.5	..	77
Myelitic, meningitic and bulbar	3	1	1	..	12
Alterations expressed in per cent.	..	7	7	..	86
Bulbar and meningitic	..	1	1	..	5
Alterations expressed in per cent.	..	14	14	..	71
Bulbar and myelitic	1	..	1	..	18
Alterations expressed in per cent.	..	..	5	..	94
Bulbar and myelitic with hydrocephalus	..	..	..	..	7
Alterations expressed in per cent.	..	..	..	..	100

* The percentages given refer to the different alterations and have no relation to the normal response. This method of expressing the relative frequency of these alterations in percentage was used because the impression is the better conveyed than by figures only.

It is most common in the types in which paralysis is not marked, as would naturally be expected. Thus, of the total alterations it comprised 20 per cent. in the meningitic cases, 17 per cent. in the ataxic, 14 per cent. in the bulbar meningitic, 11 per cent. of the myelitic. That 11 per cent. of the myelitic cases showed a diminished response is to be attributed to the character of the paralysis in these cases, the legs being affected only in a small proportion of this number and, when so, not to any marked extent.

Sluggish Reaction of the Patellar Reflex.—This is the alteration which is encountered with least frequence; it occurs, however, occasionally, and is probably more frequent than our statistical figures would show. This type of reaction is characterized by an unusual slowness and delay in response on percussion over the quadriceps tendon; we do not believe it occurs in one out of every ten meningitic cases, as our figures would tend to indicate, because a classification of only thirteen cases of this type of poliomyelitis is an insufficient number on which to base any hard and fast rules; yet we were con-

—Reflexes in the Various Types of the Disease

Left Knee Jerk					Right Plantar					Left Plantar				
Normal	Exaggerated	Diminished	Sluggish	Absent	Normal	Exaggerated	Diminished	Sluggish	Absent	Normal	Exaggerated	Diminished	Sluggish	Absent
65	10	30	4	228	144	6	16	3	85	146	4	18	3	81
..	3.5	9	1.5	84	..	5.5	14	2.5	77	..	3	17	25	76
3	2	2	1	5	5	2	..	..	2	5	2	..	..	2
..	20	20	10	50	..	50	..	..	50	..	50	..	..	50
24	9	7	..	14	33	3	4	..	3	30	2	5	..	2
..	30	23	..	47	..	30	40	..	30	..	22	55	..	22
8	..	..	..	10	11	..	..	..	6	11	..	..	..	7
..	..	..	..	100	..	..	..	..	100	..	..	..	..	100
18	11	7	3	86	56	3	9	..	35	54	2	10	..	30
..	10	6.5	2.5	80	..	6	19	..	74	..	4	23	..	72
..	2	1	..	11	5	1	..	..	5	7	1	..	..	8
..	14	7	..	78	..	16	..	..	83	..	25	..	..	75
3	1	1	..	12	5	1	1	..	7	5	1	1	..	6
..	7	7	..	86	..	11	11	..	77	..	12	12	..	75
..	2	1	..	5	6	..	..	..	1	6	..	..	..	1
..	25	12	..	62	..	..	..	..	100	..	..	..	..	100
1	..	1	..	19	10	..	..	..	6	8	..	..	1	4
..	..	5	..	95	..	..	..	..	100	..	..	..	20	80
..	..	..	..	7	1	..	..	..	5	1	..	..	..	5
..	..	..	..	100	..	..	..	..	100	..	..	..	..	100

vinced of its relative frequence where marked meningitic symptoms were present.

There was a peculiar variety of sluggish reaction which was occasionally encountered. In such instances it was necessary to percuss the patellar tendon several times before a response was obtained, and when elicited this latter was often of an exaggerated character. There appeared to be necessary a summation of impulses before the threshold of inhibition could be passed and a reaction obtained. The inflamed condition of the neuroglia tissue may account for the hyperactivity, after the threshold of inhibition was overcome.

Absence of the Patellar Reflex.—Wickman states in his monograph (p. 48) that "absence of the knee jerk is the rule."

Certainly the percentage of this alteration as compared with the percentage of other changes (Table 4) would amply justify this statement. In the purely myelitic and in the combined myelitic types, it comprised from 71 to 100 per cent. of the changes found; in bulbar types from 86 to 100 per cent. of the total alterations, while in the meningitic and ataxic forms it represented only 59 per cent. of the entire changes. These statistical findings are about what clinical observation would lead one to conclude, because it is the quadriceps group of muscles which seem remarkably prone to affection, and where the paralysis is at all marked the reflex response disappears.

"The loss of the patellar reflex may be the only demonstrable objective sign of the disease," according to Wickman, Ed. Muller and Zappert. We have encountered similar cases in which there was present only an absence of the knee jerk, and yet the spinal fluid and the history both pointed decidedly to Heine-Medin's disease. Necessarily, it is difficult to base a diagnosis on such an alteration, and it is only when an epidemic is prevalent that abortive cases of this kind are apt to be considered suspicious enough to justify the procedure of lumbar puncture. That they may occur, however, is well established, and their importance lies in the fact that although usually unrecognized they are just as dangerous in spreading the contagion of the disease as the frankly paralyzed patients.

Normal Reaction of the Patellar Reflex.—The knee jerks may remain normal throughout the paralytic phase of the disease. The bulbar and ataxic cases most commonly exhibit a normal response, representing 50 per cent. of the reflex reactions in the former and 41 per cent. in the latter (Table 3). These figures are not surprising, for paralytic involvement of the lower limbs is a negative quantity in both types. The occurrence of a normal response in 21 per cent. of the myelitic cases is to be attributed to the limitation of the paralysis in these cases to the upper extremities and trunk. It is interesting to note that in the forms of the disease in which the hydrocephalus was most marked there was a total absence of the normal response, the reflexes being altered in 100 per cent. of the cases.

The Condition of the Plantar Reflex.—The plantar reflex has been selected to show the changes that the superficial reflexes undergo, just as the patellar reflex was described to bring out the alterations occurring in the deep reflexes.

In the preparalytic stage: The plantar reflexes are exaggerated in the early part of this stage, but in a somewhat dissimilar manner to the changes that occur in the knee jerks at about the same period. The

type of reaction elicited is a peculiar example of hyperactivity, in that it probably involves mostly the sensory side of the reflex arc, and concerns a more extensive muscular response than that of a mere downward flexion of the toes. No doubt the hyperesthetic condition of the skin is the factor most concerned in producing the exaggerated response. Under normal conditions stroking the sole of the foot produces a slight flexion of the toes. In the preparalytic stage of poliomyelitis this same stimulus causes the child quickly to pull the foot out of reach of further irritation by flexing the leg on the thigh and semiflexing the thigh on the abdomen at the same time that the toes are plantar flexed.

In the paralytic stage: The reaction rapidly changes as the disease advances and becomes normal, diminished or lost by the time paralysis appears. Table 5 gives the condition of the reflex in over 600 cases in which most all of the patients were examined in the early paralytic stage.

TABLE 5.—CONDITION OF THE PLANTAR REFLEX IN THE EARLY PARALYTIC STAGE

	Right Plantar	Left Plantar
Absent	194	184
Diminished	44	50
Sluggish	6	7
Exaggerated	17	17
Altered in	261	258
Normal in	382	369
Specified in	643	627

If the figures in Table 5 are considered on a percentage basis we find that alterations were present in only 41 per cent. of the series, quite different from the condition of the knee jerks previously mentioned (Table 1), which were found altered in 81 per cent. of over 800 cases. This difference is to be attributed certainly to the fact that the plantar flexors of the toes and foot are affected much less frequently than the quadriceps group of muscles.

The relative frequence of the various alterations in the different types of the disease are given in Table 4. An exaggeration of the reflex may persist into the paralytic stage, most especially in the meningitic and ataxic cases. A diminished response was most common in the ataxic, the myelitic meningitic and the purely myelitic cases. An absence of reaction was most frequent in the purely bulbar, the combined bulbar and the myeletic types, and least frequent in the meningitic and ataxic forms.

The percentage of normal to altered reactions (Table 3) in the various forms of the disease was such that a normal response was most

common in the ataxic, bulbar, bulbar myelitic and bulbar meningitic cases.

There is more or less of a constant relation between the degree of paralysis and the presence or absence of the plantar reflex, in that when the paralytic involvement of the lower extremities is slight or partial the reflex is only occasionally missing, while when the paralysis is complete absence is the rule. In this last class of cases often the first sign of improvement is the gradual reappearance of the plantar reflex, the reaction being diminished at first, but slowly becoming normal again as the paralysis diminishes.

In cases of Heine-Medin's disease in which the polyneuritis is marked, it is easy to be deceived as to the actual extent of paralysis, especially so because one hesitates to employ passive motion of the lower limbs for this purpose, owing to the severe pain produced thereby. To avoid this the physician may be led to substitute repeated stimulations of the soles of the feet as in eliciting a plantar reflex, which would ordinarily result in the child's pulling the foot and leg, if not paralyzed, away from further irritation. But the neuritic affection renders all movement so much to be feared that the little patient will rather submit to the irritation than to move his foot or leg away, despite the fact that the degree of paralysis would in no way interfere with the motion necessary.

Ker[4] says the plantar reflex is frequently absent in epidemic meningitis. In the meningeal form of poliomyelitis we have found it absent in only 22 per cent. of the cases. From a differential standpoint, therefore, it is probably more frequently absent in epidemic meningitis than in the meningeal form of poliomyelitis.

Babinski Sign.—The pathologic alteration of the plantar reflex which is called the Babinski sign we have encountered only rarely in poliomyelitis during the paralytic stage, even in the meningeal form of the disease. Foerster,[5] however, has found it present on either one or both sides in all cases; it was not continuously demonstrable, but disappeared occasionally only to return. Wickman (p. 49) says merely that it has been observed.

In 1,017 cases terminated at the Kingston Avenue Hospital, it was encountered only 9 times in patients over 2 years of age. Kernig's sign was present in all the 9 cases; the knee jerks were absent in 5, diminished in 2, exaggerated in 1, sluggish in 1.

The type of the disease in these 9 cases was meningitic 2, bulbar myelitic meningitic 4, and meningitic myelitic 3.

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4. Ker, C. Buchanan: Infectious Diseases, London, 1909, p. 494.
5. Foerster, O.: Cited by Wickman (Footnote 2), p. 49.

Babinski's sign is quite common in tuberculous meningitis (Koplik[6] found it present in twenty-three out of fifty-two cases examined). It is not quite so frequent, but occurs in a minor percentage of cases of meningococcus meningitis. Hence its presence in a case in which the diagnosis is in question would be a point decidedly in favor of either of these diseases, especially the former, and against poliomyelitis.

The patients with poliomyelitis in which the Babinski phenomenon was found present never exhibited so marked and persistent a reaction as that seen in tuberculous meningitis.

Kernig's Sign.—In the paralytic stage we have encountered two types of response to the movements employed in testing for Kernig's sign—one, the true reaction, the other the "pseudoreaction." They are easily confused, and while the true reaction occurs only occasionally, and when so is limited to the meningitic forms of the disease, the pseudoreaction is very commonly present in cases exhibiting a moderate or severe degree of polyneuritis.

As in other forms of meningitis, the true response is characterized by a true spasm of the hamstring muscles when the thigh is semiflexed on the abdomen and an attempt is made to extend the leg on the thigh. The pseudoresponse, on the contrary, is associated with a voluntary resistance of the hamstring muscles in an effort on the part of the child to prevent further extension of the leg, owing to the pain produced. It is an analogous sign to that of Lasigne, so commonly encountered in sciatica, and the pathologic basis would seem to be somewhat similar, namely, a neuritis of the nerve trunks of the leg and thigh, the stretching of which causes intense pain. The reflex muscular spasm is lacking in these cases, and there is distinct pain on pressure over the posterior tibial nerve in the calf of the leg, over the sciatic nerve at the upper margin of the popliteal space and in the middle of the posterior aspect of the thigh.

In twenty meningeal cases Kernig's sign was mentioned as present in sixteen and was not specified in four. Statistically, it is also mentioned frequently in the combined myelitic meningitic forms of the disease, but it was a question in such cases whether to attribute it to a meningeal reaction or to a polyneuritic affection of the nerves, as often no distinction seems to have been drawn between the two.

Ankle Clonus.—Wickman and also Neurath found an ankle clonus present in cases in which an increase of the leg reflexes coexisted with a flaccid paralysis of the arm of the same side or with a lesion at a still higher level.

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6. Koplik, H.: Tuberculous Meningitis, Osler and McCrae's System of Medicine, 1913, p. 422.

Foerster[5] found a clonus in the very accute stage of the malady.

Just as in the case of Kernig's sign we had noted a true reaction and a pseudoreaction, so a similar observation was made in regard to the response obtained when passive movements were employed to determine the presence or absence of ankle clonus.

A true response was found very rare in poliomyelitis, occurring only in 6 of the 1,017 cases terminated. In these 6 cases, the type of the disease was distributed as follows: 2 were purely meningitic; 2 myelitic meningitic, 1 myelitic with facial involvement, 1 myelitic meningitic and bulbar. The paralysis was spastic in 2, and flaccid in 4. In the spastic cases both upper and lower limbs were involved; in the flaccid cases only the lower limbs. The knee jerks were exaggerated in 2 cases and absent in 3. Babinski's sign was present concomitant in 3 of the 6 cases.

A true ankle clonus is so rare in poliomyelitis as compared to the various forms of meningitis that if present in a case in which a diagnosis is in question, it would always be a point decidedly against the former disease.

The pseudoreaction is, however, frequent where polyneuritis is present, and may simulate slightly a true response; but the repeated spasmodic muscular contractions of the tendo achilles are absent and instead there is a marked voluntary resistance to the dorsal flexion of the foot, and at the same time the child screams with an intense pain which radiates up along the nerve trunks of the leg and thigh. We think that the pathologic basis for a reaction of this kind is the neuritic affection of the nerve trunks of the lower extremities, and have always found it a more or less constant evidence of the presence of the polyneuritis.

The Pupillary Reflex.—This is altered but little if at all during the acute stage of the malady. It may be slightly sluggish in cases with marked hydrocephalus, but a reaction is always present except in moribund cases. Thus, in 447 patients in which a reflex was mentioned, it was normal in 424, sluggish in 14, absent in 9. These latter 9 cases were of bulbar type, in a more or less moribund condition when examined, and no doubt the absence of the reflex was due mostly to the moribund state of the patients. We have not observed unequal degrees of reaction in the two eyes in any true case of poliomyelitis.

Patients were occasionally encountered in whom the pupils were of unequal size and in whom the reaction to light differed on either side, but a diagnosis of tuberculous or purulent meningitis was ultimately made in all such instances, either from the examination of the spinal fluid or the findings at necropsy. The pupillary reflex was tested mostly as regards light, and only occasionally as to accommodation, for,

so young were most of our patients that it was usually difficult accurately to test the reflex in this latter respect.

The Patellar Reflex in the Paralyzed as Compared to the Non-paralyzed Extremity.—In cases in which the paralytic involvement was limited to one leg, it was interesting to note the condition of the knee jerks in the affected as compared to the unaffected extremity. Draper, Peabody and Dochez[1] found the reflex present more often than absent on the healthy side. Our findings have been somewhat similar. Thus, of 23 cases (Table 6) in which the right leg alone was paralyzed, the

TABLE 6.—THE PATELLAR REFLEX WHEN ONLY ONE LEG WAS PARALYZED

Alterations	Right Leg Paralyzed		Left Leg Paralyzed	
	Right Knee Jerk	Left Knee Jerk	Right Knee Jerk	Left Knee Jerk
Normal	4	13	11	5
Exaggerated	1	1	2	1
Diminished	3	3	2	2
Sluggish	..	..	2	2
Absent	15	6	11	18
Specified in	23	23	28	28

TABLE 7.—THE CONDITION OF THE PATELLAR REFLEXES WHEN THE PARALYSIS WAS LIMITED TO THE FACIAL NERVE

Alterations	Right Facial		Left Facial	
	Right Knee Jerk	Left Knee Jerk	Right Knee Jerk	Left Knee Jerk
Normal	7	8	14	14
Sluggish	1	1	1	1
Diminished	1	1	1	1
Absent	6	5	3	3
Mentioned in	15	15	19	19

right knee jerk was absent in 15, normal in 4, exaggerated in 1, diminished in 1; while the left knee jerk was absent in 6, normal in 13, exaggerated in 1, diminished in 3. In 28 cases in which the left leg alone was paralyzed, the patellar reflex on the right side was normal in 11, exaggerated in 2, diminished in 2, sluggish in 2, absent in 11; on the left side it was normal in 5, exaggerated in 1, diminished in 2, sluggish in 2, and absent in 18.

The Condition of the Patellar Reflexes When the Paralysis Was Limited to the Upper One Half of the Body.—In 11 similar cases Draper, Peabody and Dochez found the knee jerks were present on both sides 5 times and absent 6 times. In 19 of our cases the condition of the patellar reflexes was as follows: Absent in 9 cases, diminished in 1, exaggerated in 2, and normal in 7. Such figures agree essentially with those of the authors heretofore mentioned. We noted, however, that when the paralysis was limited to one upper extremity, the knee jerks were only exceptionally absent.

The Patellar Reflexes When the Facial Nerve Alone Was Paralyzed.—In 15 cases of right facial paralysis the knee jerks were absent on the right side in 6, and on the left side in 5. In 19 cases of left facial paralysis they were absent in both legs in only 3. These figures would appear to indicate that the knee jerks may be lost in between 16 and 30 per cent. of cases of facial paralysis. The plantar reflexes remained unaltered in all instances.

SUMMARY

1. The most common symptom of poliomyelitis is an alteration in the reflexes accompanying or following a short febrile period.

2. The patellar reflex is most frequently affected owing to the common involvement of that region of the spinal cord which innervates the quadriceps extensor group of muscles. It was altered in various ways in 81 per cent. of 818 cases.

3. Hyperactivity of the knee jerks is usually, if not invariably, the first change to occur in the preparalytic stage. It may be so marked that the slightest tap with the percussion hammer will elicit a response which will cause the foot to move through an arc of from 30 to 60 degrees. The reflex, as a rule, becomes diminished and finally absent as paralysis supervenes, but it may persist exaggerated in cases in which the paralytic involvement is slight, and in which the lower extremities are not concerned to any marked extent, as well as in cases that are of the spastic variety. It may also be met with where the paralysis is limited to the upper half of the body. In rare cases there may be encountered an absence of the knee jerk on the paralyzed side and an exaggeration on the opposite and healthy side.

4. Hyperactivity of the patellar reflex is most frequent in the meningitic and ataxic cases, and in the combined types of the disease in which meningitic symptoms were prominent. Absent or normal response is the rule in purely bulbar cases, except in the very early stages.

5. An exaggerated reflex may be encountered in an atrophied and obviously paralyzed leg.

6. Not infrequently the knee jerks are found hyperactive in convalescence when every other symptom of the disease has disappeared, no doubt indicative of the stage of improvement and just preceding the return to normal.

7. A diminished patellar reflex usually precedes its disappearance with the advent of paralysis. It may persist, however, during the entire course of the acute stage, in cases with slight paralysis, especially of the meningitic and ataxic types. During the stage of improvement it is, as a rule, the first change noticed.

8. A sluggish patellar reflex is rather unusual, except in the meningeal forms of the disease. Very rarely sluggish but hyperactive reflexes are encountered.

9. Absence of the knee jerk is the most common alteration, and it is most frequently encountered in the myelitic and bulbar types of the malady. This symptom may be the only demonstrable objective sign of the disease, according to Wickman, Ed. Muller and Zappert.

10. The patellar reflex may remain normal throughout the entire paralytic phase, especially in bulbar and ataxic cases. It is also common in myelitic cases when the paralysis is limited to the upper extremities and trunk. It is scarcely ever encountered where the hydrocephalus is at all marked.

11. The plantar reflexes are exaggerated in the preparalytic stage. This hyperactivity is of a peculiar type and is accompanied by a response which comprises not only a movement of the foot, but also of the entire leg. The reflex was alterated in only 41 per cent. of 643 cases specified in the paralytic stage. An exaggerated response may persist into the paralytic stage, more especially in the meningitic and ataxic classes of cases. A diminished reaction was found most common in the ataxic, the myelitic meningitic, and the purely myelitic forms of the disease. The reflex was absent most frequently in the purely bulbar and in the combined bulbar and myelitic cases. A normal reaction was encountered most often in the ataxic, bulbar myelitic and bulbar meningitic forms of the malady. Often the first sign of improvement in cases with marked paralytic involvement is the gradual reappearance of the reflex.

12. There is to be noted two types of response to the movements employed in testing for Kernig's sign, the true reaction, the other, the "pseudoreaction" or false reaction. The true reaction is only occasionally met with, and is limited to those forms of the disease in which meningitic symptoms are extremely prominent. The pseudoreaction is common in all cases exhibiting a moderate or severe degree of polyneuritis, and is associated with voluntary resistance to extension

of the leg on the thigh, owing to the pain thereby produced. It is an analogue sign to that of Lasigne, so frequent in sciatica.

13. The Babinski phenomenon is relatively rare in poliomyelitis, but it occasionally occurs in the meningitic form of the disease. Its presence in a case in which the diagnosis was doubtful would always be decidedly in favor of tuberculous, and, to a less extent, cerebrospinal meningitis.

14. A true ankle clonus is likewise very rare in contradistinction to the other forms of meningitis. The reaction may be simulated in cases in which the polyneuritis is marked, owing to the severe pain caused by the movements employed.

15. The pupillary reflex is but little altered. It may be slightly sluggish in cases with marked hydrocephalus and during the very early stage; but a reaction is almost always present except in the moribund stage.

16. When paralysis is limited to one leg only, the knee jerks on the healthy side are more often present than absent. Also when the upper extremities are alone involved the patellar reflexes are more often present than absent. In a minor percentage of cases of facial nerve paralysis the knee jerks may be lost.

INFANTILE SCURVY

V. A STUDY OF ITS PATHOGENESIS *

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For some years we have been studying various aspects of the symptomatology and dietetics of infantile scurvy. In a recent paper[1] it was shown that pasteurized milk brings about this disorder in some cases unless an antiscorbutic food is included in the dietary. The type of scurvy induced under these conditions, as noted elsewhere, is not the textbook variety of this disease, but what has been termed latent or subacute scurvy, characterized by anemia and pallor, failure to gain in weight, rapidity of pulse and respiration, tenderness of the bones, and by a sharp recession of all these signs and symptoms when orange juice or other antiscorbutic foodstuff is given. In asserting that scurvy is brought about by pasteurized milk it should be emphasized that this is not synonymous with saying that this disorder is caused by heating the milk to the temperature of pasteurization. It is for this reason that we have, in this connection, referred to pasteurized milk rather than to pasteurization, as the former term includes not only the heating process, but the handling, subsequent cooling, aging and other factors.[2]

Some have questioned whether pasteurized milk is really involved in the production of scurvy. The fact, however, that when one gives a group of infants this food for a period of about six months, instances of scurvy occur, and that a cure is brought about when raw milk is substituted, taken in conjunction with the fact that if we feed the same number of infants on raw milk, cases of scurvy will not develop—these results seem sufficient to warrant the deduction that pasteurized milk is a causative factor. The experience in Berlin, noted by Neumann[3] and others, is most illuminating and convincing in this connection. In 1901 a large dairy in that city established a pasteurizing plant in which all milk was raised to a temperature of about 60 C. After an interval of some months infantile scurvy was reported from various sources throughout the city. Neumann[3] writes about the situation as follows:

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* Submitted for publication Sept. 7, 1917.

1. Hess, A. F.: Jour. Am. Med. Assn., 1917, **68**, 239.

2. In the article[1] referred to above on "Subacute and Latent Infantile Scurvy" this point was brought out as follows: "In referring to pasteurized milk as inducing scurvy I do not wish to state that the heating is necessarily altogether responsible for the result. There may well be other contributory factors, such as the staleness of the milk."

3. Neumann, H.: Deutsch. Klin., 1904, **7**, 341.

"Whereas Heubner, Cassel and myself had seen only thirty-two cases of scurvy from 1896 to 1900, the number of cases suddenly rose from the year 1901, so that the same observers — not to mention a great many others — treated eighty-three cases in 1901 and 1902." An investigation was made as to the cause, and the pasteurization was discontinued. The result was that the number of cases decreased just as suddenly as they had increased. It should be added that the milk was also brought to the boiling point in the home, as is the custom throughout Germany.

For two years the milk which we used at the infant asylum was pasteurized commercially at 165 F. for thirty minutes; for two subsequent years the dealers raised it to a temperature of only 145 F. for thirty minutes. According to our experience, milk heated to the higher degree of temperature induces scurvy more readily than that which is brought to only 145, judging by the results of the four-year period. During the past year we have bought raw certified milk, the best milk which is sold in the city, and pasteurized it in the institution at 145 for thirty minutes. Various formulas were prepared with this milk, so that should any disorder develop we might be in a position to analyze the trouble and correct the dietary defect. Among six infants given milk which was pasteurized and prepared for feeding on the morning it was received, none developed scurvy. One infant in our institution which had been receiving commercially pasteurized milk for many months and which showed symptoms of subacute scurvy improved on this home pasteurized milk. It may be said, therefore, that this milk manifested almost no tendency to produce scurvy. How, then, did it differ from the commercially pasteurized milk which we had previously been buying? It differed mainly, as far as we can judge, in the interval which elapsed between the heating process and the consumption of the milk. In New York City the major portion of the bottled milk is Grade B, most of which is pasteurized after it reaches the city, soon after midnight; fully two-thirds of this milk is delivered to the consumer the same morning on which it is pasteurized; however, part of it is held over and delivered twenty-four hours later. The better milk, Grade A, is largely pasteurized in the country, so that an interval of twenty-four hours elapses between the heating and delivery.[4] In order to reproduce these conditions, we held over some of the pasteurized milk for twenty-four hours on ice, so that it corresponded more closely to Grade A milk. Of eight infants who received formulas made with

4. We found that as a matter of fact the interval following pasteurization was forty-eight hours in milk which we received and which had been pasteurized at the creamery. The milk was stored by the dealers for twenty-four hours in the city, so as to provide a constant supply in case of delay or failure of delivery from the country.

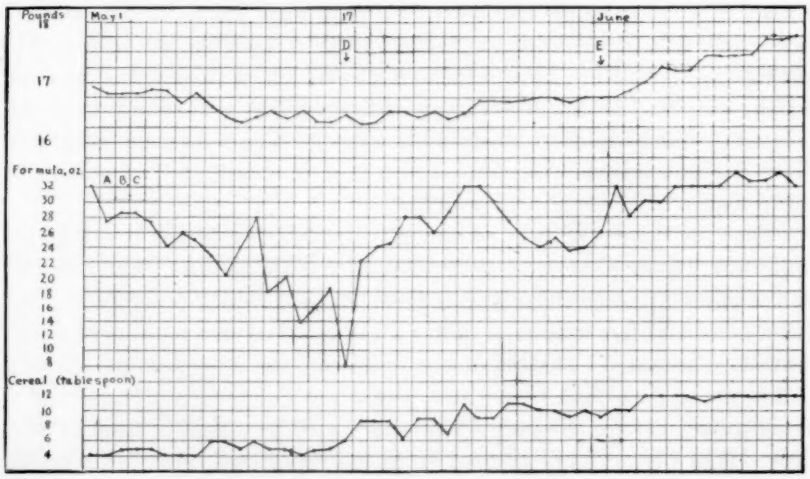
this type of milk (Pasteurized II) two showed scorbutic signs which promptly yielded to orange juice. In one of them the onset seemed to have been precipitated by an intercurrent infection of "grippe." Although these results point to the influence of the freshness or staleness of pasteurized milk, they likewise indicate that aging must be considered only a mild scorbutic agent. This deduction is forced on us by the mild nature of the scorbutic process, by the paucity of cases developing, and by the further observation that among eight other infants who were given the milk which had been kept on the ice for forty-eight hours following pasteurization (Pasteurized III), only two evinced symptoms of subacute scurvy. That aging affects raw milk similarly was shown by the fact that one baby developed latent scurvy among a group of four who were receiving Raw III milk, that is, certified milk which was kept for forty-eight hours on the ice before preparing the formula. This infant developed an eczematous skin condition, to which we have drawn attention before, and which we have frequently noted in association with scurvy. When orange juice was given the skin lesions disappeared, the general condition improved, and there was a marked gain in weight.

That the degree of heat to which the milk is subjected is not an all important factor in rendering the dietary unsuitable was clearly shown in the cases of some infants that received milk which had been boiled for a period of five minutes. After an interval of five months one well nourished infant, 11 months of age and weighing 18½ pounds, showed pallor, some periosteal tenderness, slight peridental hemorrhage and a rapid pulse. These symptoms were alleviated by a substitution of raw milk. It is furthermore evident from the reports of others that boiled milk cannot be an important etiologic factor in bringing about a scorbutic condition. When we consider the thousands of infants that receive milk of this kind and thrive, and that a moderate number must be deprived of orange juice or other antiscorbutic food, we must realize that boiled milk can lead to scurvy only in a mild degree. Statistics such as those of Variot,[5] who has distributed in his outpatient department during a period of twelve years 400,000 quarts of sterilized milk (heated in the half liter bottles and hermetically sealed at the farm) without observing a case of scurvy, must be accorded weight in this connection. It is to be noted, however, that Variot remarks that these infants frequently develop anemia unless additional diet is given, so that it must be considered open to question whether careful and repeated examinations (such as can be carried out only at home or in an institution) might not have uncovered some of the minor manifestations of scurvy. Of course, unless it is certain

5. Variot, G.: Compt. rend. de l'Acad. d. sc., 1905, **139**, 1002.

that these infants were receiving only sterilized milk, and absolutely no other food, for a period of at least six months, they cannot be considered as affording a test of the question.

Four infants were given the milk for forty-eight hours after it had been boiled, and in one instance inconclusive signs of scurvy developed. That aging, however, may also play a rôle in relation to the use of sterilized milk is well illustrated by some cases in the literature where babies developed scurvy on sterilized milk which had been sent to the country, and where they recovered on receiving the same milk freshly sterilized. In some of these instances the milk was some weeks old. There is a point, one which is lost sight of, which should be emphasized



Joseph G., aged 9 months. Chart showing stationary weight in spite of marked variation of fluid intake (due to oliguria followed by diuresis). A = Schloss milk; B = cod liver oil; C = egg yolk; D = 1 ounce of orange juice; E = potato (orange juice stopped).

in connection with a discussion of the rôle of heated milk in the causation of scurvy. It is taken for granted that if pasteurization will induce scurvy, heating the milk to a still higher degree will certainly lead to this more surely and constantly. This deduction is by no means warranted. Indeed, from personal experience, it would seem that ***milk which has been boiled is less apt to induce scurvy than milk which has been pasteurized,*** heated to 165 or a lower temperature. This was well illustrated by putting four infants, who were receiving freshly pasteurized milk, on "evaporated milk," so-called because in the course of heating for five to six hours it had been reduced to one-quarter its volume. It thus resembled in many respects the evaporated milk to be bought in the market. In preparing the formulas for these infants

due cognizance was taken of the concentration of fat, sugar and proteins of the milk. This food was well taken by the babies, and after a period of five months none manifested signs of scurvy.

As it has been claimed that scurvy is an acidosis, brought about by a long continued diet of high acidity, the reaction of the various diets was recorded. This is given in Table 1. It will be seen that pasteurized milk does not increase in acidity during the first twenty-four hours, and but little after forty-eight hours. Schloss milk, which is very prone to induce scurvy, has a very low acidity. Evidently the answer does not lie in this direction. It is probable that pasteurized milk

TABLE 1.—ACIDITY OF THE VARIOUS MILK PREPARATIONS USED

Type of Milk	Age, Hours	Number of Cubic Centimeters of Tenth-Normal Sodium Hydroxid Required to Neutralize 100 C.c. Milk							
		April 13	April 14	April 16	April 17	April 18	April 19	April 20	April 21
Raw	Fresh	14.9	14.1	16.9	18	18	17	17	18
Sterilized	Fresh	15.1	14.2	16.3	16	16	19	18	19
Sterilized (III)	48	16.0	16.4	16.5	23	21	21	23	20
Pasteurized (I)	Fresh	17.3	15.3	17.9	18	18	19	16	18
Pasteurized (II)	24	17.5	15.7	17.3	17	17	18	18	20
Pasteurized (III)	48	20.1	17.3	17.7	19	20	20	18	22
Pasteurized (III)	72*	16.6	23.2	17.0	18	27	19	21	24
Pasteurized (II) with flour†	24	....	....	....	..	13	13	14	16
Pasteurized (III) with flour†	48	....	....	....	13	36	14	19	16
Evaporated milk	24	....	....	....	..	..	29	21	21
Schloss milk	Fresh	8.4	6.4	8.7	6	7	7	6	12
Albumin milk	Fresh	26.4	28.6	41.4	40	46	34	39	49

* Last bottle of pasteurized III formula given on the following morning.
† Formula of two-thirds milk, one-third barley water, with 3 per cent. cane sugar.

constitutes quite a different medium for the growth of bacteria than raw milk, and that it may favor the development of gas-forming or other types of bacteria.

In view of the fact that it has been stated by Braddon and Cooper,[6] in connection with beriberi, that there is a definite relationship between the amount of carbohydrate in the diet and the occurrence of this disorder, for example, that when the carbohydrate is doubled, the rate of onset of beriberi is increased proportionately, it seemed worth while to observe whether this relationship obtained for scurvy. Accordingly, 3 per cent. of flour was added to the diet of four infants which were

6. Braddon, W. L., and Cooper, E. A.: Brit. Med. Jour., June 20, 1915.

receiving the usual mixture of freshly pasteurized milk and barley water. In this way a milk food was prepared similar to some of the commonly used proprietary foods.[7] No noticeable effect was observed —certainly no result which could be interpreted as indicating the existence of a biologic law such as Braddon and Cooper have formulated for beriberi, which presupposes that carbohydrate requires a proportionate amount of "vitamin" for its metabolism in the body. On the other hand, the proprietary foods, composed of carbohydrates, which are stale and have been subjected to a high degree of heat, do seem to lead to scurvy.

From the foregoing it would seem clear that the preparation and condition of the milk may lead to the development of infantile scurvy. It is equally evident, however, that the milk cannot be the sole or determining etiologic factor. The mere fact that only a few of the infants developed signs of this disorder under the various dietaries is convincing evidence of the participation of one or more additional causative agents. There is, indeed, no dietary of which it can be stated that it is certain to produce scurvy, no single food preparation of which we can postulate that, provided it is taken for a sufficiently long period of time, scurvy will certainly manifest itself. The nearest approach to this situation occurs when Keller's "malt soup" is fed to infants. It can be confidently stated that if infants are fed with this mixture of malt, flour, milk, potassium carbonate and water for five to six months, especially if pasteurized milk is used, the majority will evince definite signs of scurvy. Greatly to our surprise, we experienced this result a few years ago while we were using pasteurized milk. The fact that the disorder comes about more readily when pasteurized milk is employed, gives weight to the opinion of Neumann that the heating of milk for a second time lends it scorbutic qualities. We wish, however, to qualify his statement by adding that this seems to be true only if there is an interval between the two heating processes. If one pasteurizes the milk, allows it to cool, and then immediately brings it to the boiling point, the twofold heating does not produce the same deleterious effect.

As is well known, scurvy, infantile scurvy, beriberi, pellagra, rickets, and some other diseases have been linked together into one group; it is evident that there are symptomatic and pathologic similarities among these diseases sufficient to render it profitable to group them together. In recent papers we have drawn attention to common symptoms in infantile scurvy and beriberi, such as cardiac enlargement and tachycardia, and others have brought out points of resemblance between

7. The flour was cooked with the barley water and sugar and added to the pasteurized milk.

members of this group. The basis of the classification, however, is etiologic rather than symptomatic, and is founded on the assumption that they all are nutritional or rather dietary diseases. Although there has been an increasing tendency to admit their similarity and even their dietary nature, two totally different conceptions of their pathogenesis are current. One school regards them purely as "deficiency diseases," whereas the other believes that they are of toxic origin. Some twenty years ago Eijkmann,[8] in a paper which has become classic, described a polyneuritis which he was able to produce in fowl and which closely resembled the beriberi of man. He attributed this disorder to a toxin found in the intestinal canal, and many subsequent investigators, repeating or enlarging on his experiments, expressed themselves of the same mind. Kohlbruegge[9] was the first, however, to suggest a grouping of these diseases, designating them by the name of "fermentative diseases" and asserting that they were due to poisons which were formed by harmful bacteria which had gained predominance in the intestinal canal. Very recently Williams,[10] after many years of work in this field, has also come to the conclusion that this group of diseases is of toxic origin. On the other hand, an entirely different conception has been in vogue for the past few years, mainly due to the writings of Funk.[11] This conceives of these disorders as being due entirely to a lack of essential food substances, the "vitamins"—as being essentially deficiency diseases. A division of opinion similar to that which exists regarding the etiology of this entire group of diseases, and as to beriberi in particular, exists in regard to the pathogenesis of infantile scurvy. Barlow took the point of view that it was brought about by a diet which had been deprived of some of its essential constituents. The excellent report of the American Pediatric Society[12] cautiously refrained from expressing a decided opinion on this point, merely declaring that "the development of the disease follows in each case the prolonged employment of some diet, unsuitable to the individual child." An interesting minority report of this committee signed by Caillé, however, rendered the opinion that it is "a chronic ptomain poisoning due to the absorption of toxins." Neumann,[3] one of the most thoughtful observers and students of infantile scurvy, also regarded it as "a chronic poisoning." Hart,[13] who drew his conclusions mainly from experiments on monkeys, characterizes it as a "dyspeptic intoxication." Nevertheless, the literature contains many articles which accept Funk's

8. Eijkmann, C.: Virchows Arch. f. path. Anat., 1897, p. 253.
9. Kohlbruegge, J.: Centralbl. f. Bakteriol., Orig., 1911, **60,** 223.
10. Williams, R. R.: Am. Med., November, 1916, p. 756.
11. Funk, C.: Die Vitamine. J. F. Bergmann, Wiesbaden, 1914.
12. Rep. Am. Pediatric Soc.: Arch. Pediat., July, 1898, p. 481.
13. Hart, C.: Jahrb. f. Kinderh., 1912, **76,** 507.

point of view in its entirety and agree that we are dealing with a pure "deficiency disease."

A feature which has forced itself on the attention of many who have written on the subject is the multiplicity and variety of the foods which may bring about scurvy in the infant. Pasteurized milk, milk containing a high fat percentage, buttermilk containing but little fat, albumin milk containing much protein, and the proprietary foods rich in starches, are among the varied and incongruous number. It is of course possible that these dietaries, however different they may seem, may all be lacking a common chemical constituent. What tends strongly against this point of view is the fact, already mentioned, that with none of these dietaries can scurvy be brought about with regularity. This lack of reaction between cause and effect must lead us to conclude that the deficiency cannot be the sole cause; indeed, that it does not act directly in bringing about the disorder.

In order to gain a clearer idea as to the possible nature of the etiologic factors it will be well to turn back and consider the nature of the symptoms and pathologic changes of infantile scurvy. In the first place it should be remembered that we have signs of nerve involvement. These comprise cardiorespiratory phenomena, clearly indicating involvement of the pneumogastric nerves, occasionally changes visible in the optic disks, abnormalities of the deep reflexes, and probably sensory disturbances. The symptoms and changes common to this group of diseases, therefore, resemble those brought about by poisons of various kinds — the cottonseed poisoning in swine,[14] the toxic products of the wheat embryo,[15] or even mineral poisons such as mercurial poisoning in man.[16] The nervous symptoms, especially the irritability of the heart, may be compared to those of the enterogenous intoxication or enterotoxic polyneuritis described by Von Noorden.[17] The experiments of Abderhalden and Lampe[18] lend weight to the view of the formation of a toxin in the intestinal tract in this disorder. These investigators were able to bring about polyneuritis in pigeons and hogs by means of a restricted diet, and to cure the animals promptly by means of catharsis, castor oil or magnesium sulphate.

14. Rommel and Vedder, E.: Jour. Agric. Research, 1915, **5,** 489.

15. Hart, E. B., and McCollum, E. V.: Jour. Biol. Chem., 1914, **19,** 373.

16. In chronic mercurial poisoning the following suggestive symptoms occur: anemia, bleeding and spongy gums, loosening of the teeth, a quickened pulse, ulcers of the extremities. At times mercury attacks the nervous system, producing palsy. The resemblance is heightened by the fact that calcium metastases have been found in the muscles in experimental scurvy (Hart) comparable to those characteristic of mercurial poisoning.

17. Von Noorden, C.: Berl. klin. Wchnschr., 1913, **50,** 51.

18. Abderhalden, E., and Lampé, A. E.: Ztschr. f. d. ges. Exper. Med., 1913, **1,** 296.

They were led to attempt a cure by this means as they had noted that the polyneuritic pigeons passed very little stool and that the intestines at necropsy were markedly overfilled. They concluded that, particularly in the pig, toxic products are formed in the intestine. These novel experiments gain additional interest in view of similar results reported by McCollum[19] in connection with the scurvy of guinea-pigs. It may be inquired as to the condition of intestines in infantile scurvy. When we search the necropsy protocols for lesions of the intestinal tract we find that in most cases there are merely occasional hemorrhages and swelling of the follicles and of the neighboring mesenteric glands. In some cases there is inflammation or ulceration of the colon.[20]

If a toxin is to be regarded as the proximate cause of infantile scurvy, the question naturally arises as to the nature of the toxin.[21] Is it exogenous or endogenous? There are some sound reasons for believing that the poison generally is not introduced preformed in the food. In the first place, infantile scurvy frequently develops in babies who are given milk of the very best grade; indeed, in contradistinction to rickets, it is not preeminently a disease of the poor. Furthermore, there is no relation between the concentration of the food mixture and its liability to induce scurvy. If, among a large number of infants receiving pasteurized milk from a common source, some are given milk diluted by one-half, others given it diluted by one-third, and still others whole milk, the last group will not show a preponderating tendency to scurvy, as we should expect were the poison contained in the food. Nor is it at all uncommon to encounter scurvy in an infant which has been fed with a very dilute milk mixture. The other side of this question, however, cannot be entirely dismissed. We should bear in mind that stale pasteurized milk is more apt to produce scurvy than freshly pasteurized, a fact which might be interpreted as in favor of an exogenous toxin. Again, the reports of the scurvy of adults being occasioned by decomposed food, as in Nansen's polar expedition and as

19. McCollum, E. V.: Jour. Am. Med. Assn., 1917, **68,** 1381.

20. There is one lesion in the intestinal tract to which we would call attention, as it was present in a necropsy performed a few years ago and has been recorded by others—a marked injection of the mucous membrane of the first part of the duodenum. Additional significance would seem to be attached to this lesion in view of the fact that it is noted in some necropsy protocols in cases of beriberi and of pellagra. It may well be that this inflammation is the result of products excreted by the liver and poured into the intestine at this point.

21. There are probably many factors which influence the occurrence of scurvy. Certainly climate plays a rôle. We have frequently found that scorbutic symptoms will disappear when infants are placed out of doors and exposed to the sun and air in the spring months. Scurvy is more prevalent in the winter, as is rickets. No doubt general nutritional diseases act as predisposing factors, among which rickets and syphilis are important.

reported on ships so frequently in the past, cannot be disregarded, and seem open to the interpretation as due, in part at least, to food poisoning. The experiments of Jackson and Harley,[22] who produced scurvy in monkeys by feeding them with tainted tinned meat, would seem to fortify this interpretation. But, as we have said, infantile scurvy develops in the overwhelming majority of cases on food which is not decomposed, so that, although we acknowledge the occasional rôle of exogenous poisons, we believe that the toxin is usually elaborated within the human body. As is well known, toxins are continually being formed in the intestinal canal by bacterial growth. We are protected from the deleterious action partly by the counteraction of neutralizing bacteria. Should this protective force fail, the toxins may gain access to the tissues of the body. This is what happens in infantile scurvy. Whether the toxins differ in kind from those normally present in the intestine, or whether they are always the same, it is impossible to say. The various types of scurvy, as well as of rickets and of beriberi, may be due to differences in this regard.

According to this view, infantile scurvy is essentially a scorbutic auto-intoxication or intestinal intoxication; the diet is faulty in not being capable of inhibiting the elaboration of the poison. This raises the question, which must be left open for discussion, as to whether, from an etiologic point of view, infantile scurvy should be regarded as an entity, or whether its symptoms cannot be produced by various poisons, some exogenous and others endogenous, formed as the result of an unbalanced flora in the intestinal canal.

If we consider scurvy an intestinal intoxication, we naturally inquire as to the functioning of the bowels in this disorder. This has been frequently discussed by writers on this subject, but a consensus of opinion is not apparent. Of the cases reported by the American Pediatric Society the bowels were regular in seventy-four instances, irregular in fifteen, constipated in 126, and diarrheal in seventy-seven. This question has assumed additional interest in view of recent experimental work on animals. As has been stated, Abderhalden and Lampé,[18] as well as McCollum,[19] found marked constipation in the course of their experiments. The latter expressed his conclusions in these words: "Scurvy in guinea-pigs is the result of retention of feces. I do not know whether or not the same is true of human scurvy."

Of course, there can be no question as to whether retention of feces by itself can bring about scurvy in infants; this is excluded by the marked instances of constipation frequently encountered among thriving babies. The majority of bottle fed babies and a large number

22. Jackson, F. G., and Harley, V.: Lancet, London, 1900, p. 1184.

of the breast fed suffer from a greater or less degree of constipation. On looking up our records of infantile scurvy from this point of view, and comparing them with nonscorbutic infants, we have not been able to note a characteristic distinction. Some of the infants which developed scurvy had normal stools as far as gross examination disclosed, others suffered from constipation, while a greater number showed a record of occasional loose stools. In reviewing the cases reported by others it would seem that, as in the report of the American Pediatric Society, this same lack of uniformity is manifest, but that in the advanced cases the stools are more often diarrheal. In this connection we must bear in mind that the malt soup preparation, the diet which, in our experience, has been associated with scurvy most frequently, is essentially laxative and almost never induces constipation. And, on the other hand, the most potent antiscorbutic both for adults and for infants, one which is effective at times when fruit juices have proved unavailing, is potato, a food which has no distinct laxative properties. It may be added, as noted elsewhere, that scurvy was found to develop in infants in spite of their receiving cod liver oil or olive oil for long periods. We do not wish to conclude that the retention of feces is a negligible factor in the scurvy of infants, but rather that its rôle is quite secondary. When we assume that a poison is contained in the intestinal canal, it is evident that catharsis will be of value, and that constipation will aid in its absorption. This is true of all poisoning which occurs by way of the alimentary canal; for example, in lead poisoning, where catharsis has always been granted a distinct therapeutic place. It is probable that marked constipation may be assigned as one of the causes for the frequent occurrence of scurvy and allied intestinal intoxications among the insane, who neglect their bodily functions.

We have said that the value of potato cannot be ascribed to laxative properties. The same is true of orange juice, which is very mildly laxative, and cannot be depended on to relieve constipation in infants. Its therapeutic value in scurvy is probably due to its influence on the flora of the intestine and to its diuretic effect.

It is surprising that attention has not been called to the fact that *there is a diminished excretion of urine in infantile scurvy.* We have frequently observed this phenomenon, and its occurrence has been noted by the nurses. This oliguria is interesting from several points of view. It accounts in part for the edema which is so frequently met with in this disorder, and is of especial interest in connection with the relation of body weight to infantile scurvy. Before noting this disturbance of metabolism we were at a loss to interpret the paradoxical phenomena of a scorbutic infant which was given orange juice

being greatly improved and consuming considerable more food, but nevertheless failing to gain, or even reacting with a loss of weight. It was only when it was noted that the orange juice brought about a sudden outpouring of urine that the explanation was evident. Whereas before orange juice was given a baby urinated but three or four times a day, passing but 4 to 6 ounces of urine, thereafter it urinated fifteen to twenty times a day and passed 20 to 30 ounces of urine. A chart illustrating this condition is appended. It is probable that the citric acid of the orange juice induces the diuretic action. We had at various times tested the curative value of citric acid, considered by Netter[23] to be the essential deficiency of infantile scurvy, but, although we were able to bring about diuresis and to alleviate the symptoms temporarily, we were never able to effect a complete or permanent cure. In this connection we wish to call attention to the fact, mentioned also by Abderhalden and Lampé, that Funk's vitamins are claimed to belong to the pyridin and pyramidin groups, which possess diuretic properties. It is probable that the kidneys play a considerable rôle in infantile scurvy, its clinical course depending to some extent on the activity of their function, and that toxins are eliminated at an early stage by this route. This statement seems warranted in view of the frequency of urinary symptoms in scurvy — casts and blood cells in the urine, and the signs of parenchymatous inflammation which the kidneys may show at necropsy.[24]

Renewed interest has been awakened in the study of scurvy since it has become possible to reproduce the disease in animals. The first experimental work of this kind is generally associated with the names of Holst[25] and his co-workers, who induced scurvy in guinea-pigs by means of a diet of cereal grains. It should be noted, however, that the same result had been accomplished some years previously in this country by Theobald Smith.[26] During the past few years Baumann and Howard[27] have carried out the first metabolism investigations on scorbutic guinea-pigs. Although this work on animals has increased our knowledge considerably and is certain to open new paths of investigation, caution should be exercised in transferring the results to human beings. The fact that we are able to produce scurvy in guinea-

23. Netter, M.: Bull. Soc. pédiat., Paris, 1902, **4**, 298.

24. Pinner, F.: Deutsch. med. Wchnschr., 1896, **22**, 546.

25. Holst, H., and Froelich, D.: Ztschr. f. Hyg. u. Infektionskr., 1912, **72**.

26. In a bulletin on "Bacilli in Swine Disease," published by the Bureau of Animal Industry, 1895-1896, Theobald Smith says (p. 172): "When guinea-pigs are fed with cereals (it has been observed for some years in this laboratory), with bran and oats mixed, without any grass, clover or succulent vegetables, such as cabbage, a peculiar disease, chiefly recognizable by subcutaneous extravasation of blood, carries them off in from four to eight weeks."

27. Baumann, L., and Howard, C. P.: Am. Jour. Med. Sc., 1917, **153**, 650.

pigs by means of raw milk, a diet which effects a cure in infants, is evidence that some of the underlying conditions are different. An elaborate investigation on guinea-pig scurvy has recently appeared comprising papers by Jackson and Moody[28] and Jackson and Moore,[29] one entitled "Studies on Experimental Scurvy in Guinea-Pigs," the other "Bacteriologic Studies on Experimental Scurvy in Guinea-Pigs." The latter article is of especial interest, as it suggest tentatively that scurvy may be a bacterial infection. These investigators cultivated a diplococcus from the tissues of scorbutic animals after death, reproduced hemorrhages by inoculating cultures of these micro-organisms into the circulation, and recovered the bacteria from the tissues some weeks later. This conception of scurvy as an infectious disease has been brought forward from time to time, especially in relation to epidemics of adult scurvy. Some years ago Ausset[30] claimed to have isolated "a pasteurella type of organism" from a case of infantile scurvy, and suggested that it was the causative agent of this disease. On the other hand, Hart,[31] Rehn,[32] Hirschsprung,[33] von Starck,[34] and Schmorl[35] have failed to find bacteria in the blood, although the total number of cultures seems to have been small. Czerny[36] reports negative growth from fluid aspirated from affected joints. Jackson and Moody's results, while most interesting, are open to the criticism that bacteria were found only after death, and that all blood cultures proved negative. This is, as stated, the same experience as has been encountered in infants.

One of the most striking clinical phenomena of infantile scurvy is the marked susceptibility to infection which it entails — the frequent attacks of "grippe," the widespread occurrence of nasal diphtheria, the furunculosis of the skin, the danger of pneumonia in advanced cases. Whether this is to be in part attributed to the disturbance in water metabolism, in view of the fact that a similar susceptibility exists in *Mehlnärschaden* cases, where the tissues likewise contain an excess of water, there is no basis for judging, but the clinical fact is striking and significant.

It is as one of these secondary infections, we believe, that the findings in the guinea-pigs are to be interpreted. In this connection it is

28. Jackson, L., and Moody, A. M.: Jour. Infect. Dis., 1916, **19,** 478.
29. Jackson, L., and Moore, J. J.: Jour. Infect. Dis., 1916, **19,** 511.
30. Ausset, E.: Bull. Soc. pédiat., Paris, 1902, **4.**
31. Hart, C.: Jahrb. f. Kinderh., 1912, **76,** 506.
32. Rehn: Med. Klin., 1907, No. 30, p. 892.
33. Hirschsprung, H.: Jahrb. f. Kinderh., 1896, **41,** 1.
34. von Starck, W.: Handbuch d. Kinderh., Pfaundler and Schlossmann, 1906.
35. Schmorl: Jahrb. f. Kinderh., 1907, **65.**
36. Czerny, A., and Keller, A.: Des Kindes Ernährung, Ernährungstörungen, Leipzig, 1907, **2,** Ch. 6.

well once more to emphasize that when we refer to scurvy we do not regard it as a disease characterized by hemorrhages, but as a nutritional disorder which exists for months before the subperiosteal or other hemorrhages develop. This nutritional disorder, this simple scurvy, is not attributable to a bacterial invasion, although it leads to and is responsible for its occurrence. From the same point of view orange juice and other antiscorbutics are to be considered as of prime value in combating the toxic nutritional disturbance, but of no direct value in doing away with those hemorrhages which are brought about by bacterial infection. A hemorrhage of infectious origin, in contradistinction to those of toxic origin, must be regarded simply as a focal complication. The clinical course of many of these effusions, receding spontaneously or developing as the scorbutic condition improves, shows that they have local and not general significance. It is highly important that blood cultures should be carried out in the course of scurvy, and that particular attention should be paid to the stage of the disorder in which they are made.

Scurvy sometimes occurs in epidemic form. A few years ago we had the opportunity of observing an epidemic of infantile scurvy in connection with an outbreak of "grippe" at the infant asylum. We do not suggest this as a distinct type of this disease, for, as we have just said, infection is common as a secondary stage of scurvy. At times, however, when infection is widespread and scorbutic malnutrition exists among a group of infants, a considerable number of them may become infected and develop a hemorrhagic form of scurvy. It is an outbreak of this kind which we wish to describe in detail.

In February twelve infants in one ward developed fever and soon showed symptoms of various infections: otitis, pneumonia, nephritis, adenitis, etc. Three died of pneumonia; of the nine who recovered, seven suffered from what we shall term infectious scurvy, meaning by this a type of the disorder brought about by superimposing a secondary infection on the primary nutritional disturbance. Some of the infants showed signs of alimentary intoxication; most of them had no fever at the time the hemorrhages occurred, although they may have had a rise of temperature when the infection began. In April a second epidemic of "grippe" took place and three more infants developed scurvy. The signs were mainly hemorrhagic, developing at sites both atypical and typical for scurvy. They occurred as follows: Case 1, left diaphysis of humerus and tibia; face; anterior abdominal wall at site of serum injection; right eyelid; Case 2, abdominal wall; cranium; vertebral column; external ear; Case 3, diaphysis of left femur; Case 4, cranial bones and external ear; Case 5, external ear and face; Case 6, diaphysis of tibia; Case 7, abdomen (Table 2).

These infants were receiving formulas made from milk pasteurized at 160 F. for twenty minutes.

It is striking that many of the hemorrhagic signs above outlined are quite different from those encountered in infantile scurvy. We have never met with such widespread subcutaneous hemorrhages, and have noted them in the literature only in the most advanced cases. But what makes these cases stand out sharply from ordinary scurvy is not

TABLE 2.—DATA OF EPIDEMIC OF SCURVY

Case	Age, Mos.	Weight Lbs.	Weight Oz.	Site of Hemorrhages	Date	Diet	Remarks
1 J. H.	6½	9	6	Humerus, tibia, face	Apr. 19	Breast milk (1 week); breast milk, buttermilk and orange juice	Grippe since end of February; nephritis; v. Pirquet negative
		10	4	Upper eyelid.....	May 9		
2. L. S.	5	12	14	External ear, parietal bones, vertebral column, abdominal wall	May 4	Pasteurized milk formula; orange juice 1 oz. daily since April 22	Twitchings and convulsions; signs of intoxication; red blood cells in urine; fever to 101 F.; v. Pirquet negative
3. A. R.	10	12	8	Femur.....	Apr. 19	Pasteurized milk formula; vegetables for a month; orange juice longer; getting orange juice and vegetables	Grippe end of January; again in April; fever until April 17; v. Pirquet positive; gums negative
				Femur again swollen and tender	June 4		
4. D. E.	5	7		Both ears; parietal bones	Apr. 29	Breast milk since April 19; May 30, changed to pasteurized milk	Grippe throughout March; intoxication; nephritis; no relapse although no orange juice given
5. T. K.	10	15	13	Ear and face.....	Apr. 27	Pasteurized milk, cereal, vegetable, soup; orange juice since April 15	Two teeth; gums negative; v. Pirquet negative
6, P. G.	4	8	4	Tibia.....	May 8	Pasteurized milk formula	Grippe end of February and first half of March; gained 20 oz. during last month; v. Pirquet negative
7. I. P.	2	6	4	Abdomen.....	Mar. 7	Breast milk for past week	Grippe; probable source of epidemic

so much their exceptional symptomatology as their lack of response to specific therapy. Two had received breast milk for two weeks previous to the onset of symptoms; one an ounce of orange juice for two weeks; one an ounce of orange juice and also vegetable for two months previously; one vegetable and orange for a week; and another vegetable for an indefinite period before the onset. One baby, 9 months old, which had a tender swelling of the diaphysis of the femur in April, had a relapse of symptoms in June in spite of having received

orange juice and vegetables throughout the intervening period. It should be noted that of the seven infants four were under 6 months of age, which, from a practical standpoint, and although subject to exceptions, is rightly regarded as the minimum age for the hemorrhagic stage of this disorder. Again, although two babies had teeth, neither showed spongy gums nor peridental hemorrhage. The swellings of the long bones, however, were quite typical of the subperiosteal hemorrhages of infantile scurvy, and are open to no other clinical diagnosis.

How are we to interpret this picture? The ages of the infants, the distribution of the hemorrhages, the development of signs in some cases in spite of antiscorbutic diet, the sharply defined epidemic character of the disease, distinguish it from the scurvy we commonly meet with. We believe that the epidemic was brought about by a bacterial invasion. Whereas usually scorbutic intoxication must be prolonged for a period of about six months to allow the secondary invasion of bacteria, in this instance, due probably to the peculiar nature of the bacteria, invasion occurred early and in some infants which were receiving an antiscorbutic diet. In most of these instances, however, it should be noted that the infants had been receiving pasteurized milk for months, and the antiscorbutic foodstuff had been added but a few weeks previously. Latent scurvy was prematurely changed to florid scurvy by the presence of a ward infection; an epidemic of "grippe" precipitated an epidemic of scurvy exceptional in its hemorrhagic tendency.

In closing we may add that this epidemic resembles the epidemics of melena neonatorum which occur from time to time in lying-in hospitals and which formerly visited these institutions so frequently. Not long ago we had the opportunity of observing an epidemic of this character where, within a few weeks, some eighteen new-born infants developed hemorrhages at various sites — the umbilical cord, mouth, nose, skin, etc. These cases must also be considered bacterial in nature. Finally, we wish to suggest that many of the reported instances of scurvy in breast fed babies are to be regarded as cases of sepsis grafted on general malnutrition due to syphilis, tuberculosis and undernourishment, rather than as true scurvy.

CONCLUSIONS

One of the several factors in the pathogenesis of infantile scurvy is faulty diet. Pasteurized milk was found to be a contributing cause if it was not fresh — if given twenty-four to forty-eight hours after pasteurization. From this point of view milk pasteurized in the city is preferable to milk pasteurized at the creamery, which reaches the consumer much longer after the heating process. Aging seemed to

play a greater rôle in the production of scurvy than heating, whether the milk was pasteurized or raised to the boiling point. It was found that even raw milk on aging loses its antiscorbutic properties.

Infantile scurvy is not, however, a simple dietary disease. The diet is at fault in allowing the intestinal bacteria to elaborate toxins. It is doubtful whether the toxin is always the same, and therefore whether, from a strictly etiologic standpoint, this disorder should be regarded as an entity. Infantile scurvy is an intestinal intoxication or an autointoxication due to the overgrowth of harmful bacteria in the intestine. It is the product of an unbalanced flora which is no longer controlled by a proper dietary.

Oliguria is a common symptom of scurvy. The mild therapeutic effect of citric acid may be ascribed partly to its diuretic properties. Orange juice also was found to bring about marked diuresis.

One of the striking and important symptoms of scurvy is a susceptibility to infection (furunculosis, nasal diphtheria, "grippe," etc.). Some hemorrhages are due to this secondary infection, and are to be regarded not as scorbutic, but rather as focal complications. Other hemorrhages are truly scorbutic. Scurvy, however, is essentially a disorder characterized by malnutrition and not by hemorrhage, taking months to develop, and, from a clinical point of view, frequently latent or subacute.

Infantile scurvy occurring in epidemic form is described. This results when latent scurvy exists among a number of infants and an infectious disease (such as "grippe") is superadded.

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SOME OBSERVATIONS ON INTESTINAL BACTERIA IN CHILDREN *

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During the past few years there has been a great revival of interest in the bacteriology of the digestive tract. The investigations of Moro,[1] Tissier[2] and others, especially Herter and Kendall[3] in this country, have explained the discrepancy noted since the days of Escherich, that the number of bacteria in the dejecta is much greater than the number of colonies which develop on our plates. They have demonstrated that the gram-positive organisms found in smears of the intestinal contents are equally important with the gram-negative, that they can be freed from the ordinary intestinal bacteria by cultivation in acid broth, and that they are just as much obligate inhabitants of the alimentary canal as are *Bacillus coli communis* and *Bacterium lactis aerogenes*. The old theory that *Bacillus coli* is gram-positive in the intestines and gram-negative in our cultures was thus proved to be untenable, as was usually suspected. Along with this development of our knowledge, Metschnikoff[4] and his followers have attempted to bring about a change in the character of the bacteria in the digestive tract by the administration of food materials containing bacteria producing large quantities of acid from lactose. It was claimed that the ingested bacteria, particularly the so-called Bulgarian bacilli, would overcome and displace the species previously multiplying in the intestine, *Bacillus coli* and the putrefactive anaërobes. Despite the somewhat extravagant claims of this school of investigators, it has not yet been clearly shown that the ingested organisms are definitely implanted on the intestinal mucosa in man, although Rettger's experiments indicate that such a transplantation may occur in small animals.[5] Furthermore, it should also be indicated that the poisonous action of the putrefactive bacteria in the intestine, while possibly evident clinically in selected cases, has not yet

* Submitted for publication Aug. 29, 1917.

* From the Bacteriological Laboratory and the Harriet Lane Home, Johns Hopkins University.

1. Moro: Wien. klin. Wchnschr., 1900, **13,** 114.

2. Tissier: Recherches sur la flore intestinale, normale et pathologique du nourrison, 1900.

3. Herter and Kendall: Jour. Biol. Chem., 1910, **1,** 203. Kendall: Ibid., 1909, **6,** 268.

4. Metschnikoff: The Prolongation of Life, Bacteriotherapie.

5. Hull and Rettger: Jour. Bacteriology, 1917, **2,** 47.

been clearly shown experimentally. Finally, there is a good deal of evidence to show that the changes which do occur after the administration of food materials containing Bulgarian bacteria may be due to the chemical constituents of the food, the lactose and the lactic acid, and not due to the bacteria themselves. Nevertheless, the idea that profound changes may occur in the bacteria of the intestinal canal as the cause of pathologic conditions, or as their accompaniment, and that these changes, particularly the development of a gram-positive in place of a gram-negative flora, may be of diagnostic significance and point the way to remedial treatment, has become firmly intrenched in the minds of clinicians. This is not without reason in view of the facts which have been brought out recently. There are, however, many contradictions in the published results, and it may be doubted whether our knowledge of the conditions which must exist in such a large and varied area for bacterial development as the gastric and intestinal mucosa is yet sufficiently accurate to permit any exhaustive generalization which may be applied clinically. In view of these considerations it has seemed to us worth while to take up certain phases of the problem again, and in the present investigation we have limited ourselves to one particular inquiry, namely, to what extent differences in the bacterial flora of children can be correlated with the type of food forming the sole or the chief ingredient of the diet. For the solution of this problem we have been allowed to utilize the dejecta of the breast-fed infants in the maternity ward of the Johns Hopkins Hospital and those of the young children in the Harriet Lane Home.

In the early part of our studies we made routine examinations of about fifty children fed on mother's milk, by the use of the ordinary mediums, such as plain and dextrose agar and dextrose broth in the fermentation tube. As might have been expected, we obtained in these cultures practically nothing but gram-negative bacteria and no information of value was elicited. It was then decided to make an intensive study of a few cases by a careful microscopic examination of the dejecta and by the adoption of special methods for the cultivation of the various kinds of the micro-organisms seen in the smears. It is the results of the examination which now we wish to report.

Smears from the evacuations of children when stained by gentian violet, methylene blue and by Gram's method reveal six different types of organisms as follows:

1. Small gram-negative bacilli.
2. Small gram-positive bacilli.
3. Gram-positive micrococci and streptococci.
4. Gram-positive yeast cells.
5. Large gram-positive bacilli.
6. Spores of various sizes and shapes.

All these various types of organisms can be cultivated by appropriate methods. With ordinary plates, especially if sugar be present in the medium, the gram-negative bacilli tend to overgrow and crowd out the other species, and one finds *Bacillus coli, Bacterium lactis aerogenes, Bacillus alkaligenes* and occasionally some of the rarer liquefiers of the intestines. The small gram-positive bacilli are chiefly aciduric organisms. They do not appear on agar plates, but may be cultivated on acid broth (2 to 3 per cent.) which inhibits the other species. After two or three transfers in this medium, which suffices usually to effect a separation, these aciduric organisms can be obtained in pure culture on agar or blood serum. They grow well on acid agar and in our experience grow best on acid whey agar. This group evidently represents the aciduric organisms of Moro, *Bacillus acidophilus,* although at times we have encountered species which resemble those described by Heinemann and which he puts provisionally in the group of Bulgarian bacilli. The yeast cells seldom appear on plain agar plates. They grow out abundantly in acid broth with the aciduric bacilli, but can also be obtained on acid whey agar plates. They grow best on dextrose agar plates made directly from the dejecta, where they appear after the lapse of six to ten days, when the agar has become highly acid from the growth of *Bacillus coli.* In such old plates the large viscid colonies of the yeast cells are easily recognized. The greatest interest attaches to the larger gram-positive bacilli of the intestines. They are of various sizes, sometimes small, measuring 0.5 to 0.75 by 2 to 3 microns, sometimes large, measuring 1 to 1.5 by 3 to 8 microns. Their protoplasm may be regular as in the smaller forms, or highly irregular as in the large forms, which seem to be made up of spherical bodies. These organisms appear occasionally on agar plates. They can best be cultivated by heating portions of the dejecta in broth to 85 C. for ten minutes, pouring plates at once, and after twenty-four hours' incubation of this broth. By such a procedure an abundant growth of organisms can be obtained which can now be identified as the aerobic spore-bearing bacteria found in soil, dust, water and milk. In plates poured directly these species have the same morphology as that observed in the smears, and it is sometimes possible, on bacterioscopic examination, to give a provisional opinion as to the species which can be cultivated. This is true especially when the large granular bacilli of the megatherium-petasites group are found. The species cultivated from heated material, immediately and after incubation, are much more varied. The size and shape of the spores produced by these organisms are quite characteristic and can usually be correlated with the spores found in the smears.

We have examined the spore-bearing bacteria on plates of the dejecta in about fifty cases, and we are of the opinion that they repre-

sent the large gram-positive bacteria and the spores found in smears. We see no reason to believe that these gram-positive organisms are anaerobes. The resemblance in morphology of the aerobic spore-bearers cultivated, both in the vegetative state and in the spore state, to the vegetative rods and the spores found in smears is so marked as to lead to the opinion that they are the same. We do not deny the possibility that anaerobic bacteria are present in the intestinal tract in probably all cases. We believe, however, that the majority of the gram-positive rods and the spores represent aerobic species. Of special interest is the occasional presence of a small organism with a round terminal spore looking not unlike the tetanus bacillus. The finding of this organism is sometimes regarded as indicative of intestinal putrefaction, since it has been identified on the basis of its morphology alone as *Bacillus putrificus coli* of Bienstock. This organism can be cultivated without difficulty and can now be recognized as one of the common aerobic spore-bearers, *Bacillus pseudotetanus*.

We have been able to examine somewhat exhaustively the dejecta of ten breast fed babies and of eighteen artificially fed children, varying in age from 1 to 10 years. These latter were fed on a mixed diet, on raw and boiled cow's milk, on buttermilk or on protein milk. The gram-negative bacilli have shown no variation in the different cases. Except in rare instances, they are the most abundant of the species present and they can always be cultivated on agar plates. The aciduric bacilli were obtained in seven of the ten breast fed babies and in all of the eighteen children. There was no particular predominance of these organisms except in one case, and their presence although in small numbers in all of the artificially fed children and in 70 per cent. of the breast fed infants suggests that they could always be found were the search extended. In one instance the stool of a normal suckling was made up almost exclusively of long gram-positive bacilli which could not be cultivated by any method tried. Branching aciduric organisms of the type of *Bacillus bifidus* were not encountered in our series either in the breast fed or in the artificially fed children. Gram-positive micrococci and streptococci were found in six of the ten babies and in ten of the eighteen artificially fed children. Many of these were distinctly aciduric; others, however, were streptococci which grow on ordinary plates, while others were evidently *Micrococcus ovalis*. As far as could be learned, no significance is to be attached to the presence of organisms of this group, but the fact that they were isolated from 60 per cent. of cases leads to the opinion that like the aciduric bacilli they are obligate inhabitants of the intestinal canal. Yeasts were obtained in six of the eighteen children. In two of these cases the children were fed on buttermilk, which on culture proved to contain yeasts in great numbers. In three other cases milk was the chief

article of diet and in one instance the child was being nourished on protein milk. In one case the stool of a six weeks' baby suffering from pyloric stenosis and fed on breast milk was also full of yeasts.

In the dejecta of the breast fed children aerobic spore-bearing bacteria were obtained in all but one case. They were present in considerable variety in the original smears, even in the youngest nurslings, evidently finding their way into the alimentary canal of the child as soon as it begins to swallow. They were not so abundant as to grow out on plates made directly from the dejecta or to any extent from heated material before its incubation. They could, however, always be obtained from this material after incubation for twenty-four hours. In two cases the spore-bearing bacteria were markedly increased in these breast fed children. They appeared in great profusion on our original plates and in plates poured directly from heated material. In one of these cases the stool was made up largely of meconium mixed with a soap suppository and in the other case the stool was very scanty and material for cultures had to be scraped from the napkin. We do not, therefore, regard these cases as indicative of any real increase of spore-bearers in the intestinal tract. The spore-bearers found in breast fed children were chiefly those species which have now been shown to be the prevalent bacteria of our environment. *Bacillus cereus* and *Bacillus albolactus* were the most frequent isolations, *Bacillus pseudotetanus* and *Bacillus mesentericus* were somewhat less common, while *Bacillus subtilis, Bacillus petasites* and *Bacillus vulgatus* were among the rarer findings. This relative proportion of the species is about the same as the proportion established for soil, dust and water in Baltimore in which *Bacillus cereus* and *Bacillus albolactus* are the most abundant.[6] It is interesting to note that often the same organisms were isolated from different patients on the same day, pointing to some sort of a ward infection as their source.

In the artificially nourished children aerobic spore-bearers were found in smears from the dejecta in all instances. In three cases they were not isolated from plates made from either the raw or heated material. One of these was a child with pyloric stenosis fed on breast milk and formula, another was a child with pneumonia fed on mother's milk and barley water, and the third was a child with ileocolitis fed on protein milk and formula. The aerobic spore-bearers were cultivated in 15 of the 18 cases. Practically the same varieties were found as in the breast fed children, *Bacillus cereus* and *Bacillus albolactus* being the most common. Pseudotetanus bacilli and members of the megaterium and mesentericus groups were also isolated. There was an increase of gram-positive aerobic spore-bearers in ten of the 18 cases.

6. Lawrence and Ford: Jour. Bacteriology, 1916, **1**, 273. Laubach: Jour. Bacteriology, 1916, **1**, 493.

The organisms of this character were greatly increased in numbers in the smears, and cultures showed them in abundance in the heated and incubated material. In 6 of these cases these species were so abundant as to appear in large numbers on the plates poured directly from the dejecta. In 8 of these 10 cases the children were fed on protein milk; in 5 of these the stools were normal, in 1 case constipated, in 1 case semisolid and in 1 case diarrheal. Of the 2 other cases with an increase in spore-bearers, one child was suffering from diarrhea and was being fed on buttermilk; the other was a feeding case with normal stools. From this patient material was examined on four different occasions. No increase in spore-bearers was detected on the first three examinations, at which time the patient was being nourished on whole milk, barley water and sugar. Subsequently, wheat flour and malt soup were added to this diet, and the next examination showed an increase of spore-bearing bacteria. Eight of the artificially fed children failed to show any increase of large gram-positive bacteria. One of these patients was fed on protein milk and one on buttermilk. The others were receiving either a general diet, breast milk mixed with barley water, or cow's milk and barley water. We thus see that, with two exceptions, an increase of aerobic, spore-bearing bacteria in the intestinal discharges, gram-positive in character, is regularly associated with a diet of protein milk or buttermilk.

The question now arises as to the explanation of this association, which we believe to be quite definite. Does the diet of protein milk or buttermilk, for instance, so change the character of the intestinal contents as to lead to profound alterations in the conditions for bacterial development, or is there a possibility that these organisms make their way into the alimentary canal with the food and find there either a temporary or a permanent nidus for development? We believe this latter explanation to be the correct one. To throw some light on this question a bacterologic examination of the various foods employed in the Harriet Lane Home was undertaken, including pasteurized milk, protein milk, buttermilk and farina. All these articles of diet were found to be infected with aerobic, spore-bearing bacteria, the spores of which are capable of withstanding the heat employed in ordinary cooking. The bacteria found in protein milk and in buttermilk were especially abundant, *Bacillus cereus* and *Bacillus albolactus* being the predominant types. In the heated milk they were somewhat less common, the same species predominating. Farina was relatively free from bacteria and in the samples examined contained chiefly pseudotetanus bacilli. The various articles of diet contained the following species of bacteria:

Protein Milk: *Bacillus cereus, Bacillus albolactus, Bacillus mesentericus, Streptococci,* molds.

Heated Milk: *Bacillus cereus, Bacillus albolactus, Bacillus mesentericus,* micrococci, molds.

Buttermilk: *Bacillus cereus, Bacillus albolactus, Bacillus mesentericus,* yeasts, molds.

Farina: *Bacillus pseudotetanus, Bacillus cereus, Bacillus albolactus, Bacillus mesentericus,* molds.

From the results of this examination it may be seen that the bacteria with which the food is infected are almost the same as those found in the dejecta of the children fed on these foods. *Bacillus cereus* and *Bacillus albolactus* are the most common, while *Bacillus mesentericus* and *Bacillus pseudotetanus* also appear. The relative proportion of the species encountered in the food is also strikingly similar to that established for the dejecta. We are, therefore, inclined to the opinion that the presence of aerobic spore-bearing bacteria, gram-positive in character in the dejecta of children, is to be attributed to their mechanical introduction with the richly infected food. This we believe particularly striking in the cases in which the children are fed on protein milk and buttermilk. That a change in the chemical ingredients of the food employed may be the causative factor in the appearance of the large gram-positive bacteria in the dejecta is not ruled out by our methods of investigation and for the present our conclusion cannot be regarded as anything more than a well defined opinion. We hope to clear up this point in future studies. We see no reason to believe that the presence of large gram-positive organisms is indicative of anaerobic putrefaction, however, nor is it of any diagnostic value, since it occurs with a variety of clinical conditions. It is possible that we have here an actual substitution of bacterial species in the intestine, but whether this substitution is temporary or permanent is not clear from the work thus far completed. It is interesting to note that the results of this work lend no support to the old contention of Flügge[7] that the resistant spore-bearing organisms which survive in boiled milk may pass through the acid gastric juice, multiply in the alkaline contents of the lower bowel and by their toxic products set up diarrheal conditions. The presence of normal, constipated or loose stools in cases in which there is a great increase in aerobic spore-bearers indicates that these organisms may be regarded as practically harmless in the alimentary canal, even if they be present there in large numbers. Finally, our results indicate that one should be extremely cautious in drawing any conclusions which have a clinical bearing from a bacteriologic examination of the dejecta unless a study of the bacteria in the ingested food be made at the same time.

7. Flügge: Ztschr. f. Hyg., 1894, **17,** 272.

CASES EXAMINED

BREAST-FED CHILDREN

CASE 1.—H., 3 to 4 days; stool normal. Smears showed gram-negative bacilli, gram-positive cocci and a few large gram-positive bacilli. Cultures revealed *Bacillus coli*, aciduric cocci and four spore-bearers in small numbers, *Bacillus cereus*, *Bacillus pseudotetanus*, *Bacillus mesentericus* and *Bacillus petasites*. No increase of spore-bearers.

CASE 2.—J., 4 days; stool normal. Smears showed gram-negative bacilli, small gram-positive bacilli and cocci and large gram-positive bacilli and oval spores. Cultures revealed *Bacillus coli*, *Bacillus cereus* and *Bacillus albolactus* in considerable numbers. This stool was small, chiefly meconium, and in making the cultures the material had to be scraped from the napkin. The increase of spore-bearers may be explained on this basis.

CASE 3.—P., 7 days; stool normal. Smears showed chiefly gram-negative bacilli, gram-positive cocci and large gram-positive bacilli with some terminal spored organisms. Cultures revealed *Bacillus coli*, aciduric bacilli and *Bacillus cereus* in great numbers. This stool was obtained by means of a soap suppository, and the increase of spore-bearers may have been due to this factor.

CASE 4.—P., 15 days; stool normal. Smears showed gram-negative bacilli and a few large gram-positive bacilli. Cultures gave *Bacillus coli* and *Bacillus cereus*. No increase of spore-bearers.

CASE 5.—B., 5 days; stool normal. Smears showed gram-negative bacilli, small gram-positive bacilli, large gram-positive bacilli and large cocci. Cultures revealed *Bacillus coli*, aciduric cocci, *Bacillus cereus* and *Bacillus mesentericus*. No increase of spore-bearers.

CASE 6.—K., 12 days; stool normal. Smears showed gram-negative bacilli and small gram-positive bacilli. Cultures revealed *Bacillus coli*, aciduric bacilli and cocci, *Bacillus pseudotetanus* and *Bacillus petasites*. No increase of spore-bearers.

CASE 7.—G., 5 weeks; stool normal. Smears showed gram-negative bacilli, small gram-positive bacilli and cocci. Cultures showed *Bacillus coli*, aciduric bacilli and cocci, *Bacillus cereus* and *Bacillus pseudotetanus*. No increase in spore-bearers.

CASE 8.—W., 19 days; stool normal. Smears showed gram-negative bacilli, gram-positive cocci. Cultures revealed *Bacillus coli*, aciduric bacilli and cocci and *Bacillus pseudotetanus*. No increase of spore-bearers.

CASE 9.—M., 8 months; stool normal. Smears showed gram-negative bacilli, small gram-positive bacilli and large gram-positive bacilli. Cultures revealed *Bacillus coli*, aciduric bacilli and cocci and a variety of spore-bearers in small numbers, *Bacillus cereus*, *Bacillus pseudotetanus*, *Bacillus vulgaris* and *Bacillus mesentericus*. No increase of spore-bearers.

CASE 10.—R., 6 weeks (Harriet Lane Home). Pyloric stenosis; stool normal; breast fed. Smears showed very few gram-positive bacilli, many thin, rather long gram-positive bacilli, a typical gram-positive flora. No large gram-positive organisms were found. Cultures revealed aciduric cocci and aciduric bacilli of a different morphology from that of the organisms seen in the smear. Yeast cultivated. No spore-bearers found.

ARTIFICIALLY FED CHILDREN

CASE 11.—N. S., 3 months (H. L. H.). Feeding case; stool normal. Diet, whole milk, barley water and cane sugar. Stools examined on four occasions. Smears showed small gram-negative bacilli, small gram-positive bacilli and micrococci, large gram-positive bacilli and terminal spored organisms. Cultures showed *Bacillus coli*, aciduric bacilli and micrococci and yeasts, together with

Bacillus cereus, Bacillus albolactus and *Bacillus pseudotetanus.* No increase of spore-bearers. In this case there was a good deal of variation in the amount of sugar in the diet, and wheat flour and malt soup were added to the whole milk and barley water, after which the spore-bearers were definitely increased.

CASE 12.—B., 6 weeks (H. L. H.). Diarrhea; stools numerous and watery. Diet, breast milk and buttermilk. Smears from the stool showed gram-negative bacilli, small gram-positive bacilli and large gram-positive bacilli. Cultures showed *Bacillus coli,* aciduric bacilli, and *Bacillus cereus.* Spore-bearers increased.

CASE 13.—K., 4 years (H. L. H.). Intestinal indigestion; stool large, semi-solid, foul, pasty and full of fat and undigested meat fibers. Diet, protein milk and junket. Two examinations. Smears showed gram-negative bacilli, small gram-positive bacilli, large gram-positive cocci, large gram-positive bacilli. Cultures showed *Bacillus coli,* aciduric bacilli and micrococci, and *Bacillus cereus.* No increase of spore-bearers in the first examination on admission to the hospital. On second examination, after a diet of protein milk and junket was established, the spore-bearers were definitely increased.

CASE 14.—P., 3 months (H. L. H.). Fatal diarrhea; stools numerous, watery, containing a small amount of mucus and numerous curds, varying in size from that of a small pea to that of a small lima bean. The vomitus contained the same curds. Diet, protein milk and buttermilk. Stools examined three times. Smears showed gram-negative bacilli, small gram-positive bacilli and cocci, large gram-positive bacilli. Cultures revealed *Bacillus coli,* aciduric bacilli and micrococci, and yeasts, together with *Bacillus cereus, Bacillus albolactus* and *Bacillus mesentericus.* Spore-bearers increased. The curds showed a few gram-negative bacilli, many gram-positive cocci and yeasts and a great predominance of gram-positive bacilli. Cultures from them revealed yeasts and the three species mentioned above in overwhelming numbers. Their presence in the vegetative state in the curds is suggestive of an actual multiplication in the passage of the curds through the alimentary canal.

CASE 15.—H., 2 years (H. L. H.). Acidosis; stools frequent, foul, watery and bloody. Diet, buttermilk and farina. Two examinations. Smears showed gram-negative bacilli, small gram-positive bacilli and micrococci, large gram-positive bacilli, yeasts and spores. Cultures revealed *Bacillus coli,* aciduric bacilli, yeasts, *Bacillus cereus, Bacillus albolactus* and *Bacillus mesentericus.* No increase of spore-bearers.

CASE 16.—D. (H. L. H.). Fatal acidosis; stools numerous and watery. Diet general and restricted. Smears showed gram-negative bacilli, small gram-positive bacilli and cocci, large gram-positive bacilli, yeasts, oval spores and terminal-spored bacilli. Cultures revealed *Bacillus coli,* aciduric bacilli, together with *Bacillus cereus, Bacillus subtilis* and *Bacillus albolactus.* No increase of spore-bearers.

CASE 17.—W., 1 year (H. L. H.). Pneumonia; stools large and constipated. Diet, heated milk, barley water, farina and orange juice. Smears showed gram-negative bacilli, small gram-positive bacilli and cocci, large gram-positive bacilli, spores and yeasts. Cultures revealed *Bacillus coli, Bacillus lactis aerogenes,* aciduric bacilli and cocci, yeasts and *Bacillus cereus.* No increase of spore-bearers.

CASE 18.—W., 7 months (H. L. H.). Pneumonia; stools frequent, watery. Diet, protein milk. Smears showed gram-negative bacilli, gram-positive cocci, large gram-positive bacilli. Cultures revealed *Bacillus coli, Bacillus lactis aerogenes,* aciduric bacilli and cocci, together with *Bacillus cereus.* No increase of spore-bearers.

CASE 19.—B. W., 5 months (H. L. H.). Eczema; stool normal. Diet, milk, barley water, sugar and farina. Smears showed gram-negative bacilli, small gram-positive bacilli and cocci and large gram-positive bacilli. Cultures revealed

Bacillus coli, aciduric bacilli and cocci, yeasts, together with *Bacillus cereus* and *Bacillus pseudotetanus.* No increase of spore-bearers.

CASE 20.—L., 8 months (H. L. H.). Feeding case after diarrhea; stools normal. Diet, protein milk and buttermilk. Smears showed gram-negative bacilli, small gram-positive bacilli and large gram-positive bacilli. Cultures revealed *Bacillus coli,* aciduric bacilli and cocci, together with *Bacillus cereus, Bacillus albolactus* and *Bacillus pseudotetanus.* Spore-bearers increased.

CASE 21.—H., 3 months (H. L. H.). Diet, protein milk. Smears showed gram-negative bacilli, small gram-positive bacilli and cocci, large gram-positive bacilli. Cultures revealed *Bacillus coli,* aciduric bacilli, yeasts and several of the common spore-bearers. Spore-bearers increased.

CASE 22.—B., 10 months (H. L. H.). Acidosis; stools large and constipated. Diet, protein milk and dextromaltose. Smears showed small and medium sized gram-negative bacilli, gram-positive diplococci or short streptococci, small gram-positive bacilli and large gram-positive bacilli in considerable numbers. These last were of various sizes and shapes. Some had round ends, others square ends and many showed degenerated cytoplasm. A few very large gram-positive organisms were found with large subterminal spores. Small spores of the subtilis or mesentericus type were also found. Cultures revealed *Bacillus coli,* aciduric bacilli, *Micrococcus ovalis* and *Bacillus subtilis.* The plates from the raw material showed no increase of spore-bearers. Plates from the heated material before and after incubation showed many colonies of *Bacillus subtilis.*

CASE 23.—W., 8 months (H. L. H.). Acidosis and polyuria; stools normal. Diet, protein milk and dextromaltose. Smears showed small and medium sized gram-negative bacilli, small gram-positive bacilli, small and large gram-positive diplococci and a great increase of large gram-positive bacilli. These last were of various sizes and shapes, some with round ends, others with square ends resembling *Bacillus cereus* or *Bacillus megaterium* in morphology. Cultures revealed *Bacillus coli, Micrococcus ovalis,* aciduric bacilli and cocci, and *Bacillus subtilis.* No increase of spore-bearers on plates made from raw material. *Bacillus subtilis* in great profusion on plates made from heated material, both before and after incubation.

CASE 24.—A., 7 months (H. L. H.). Pneumonia; stools constipated. Diet, breast milk and barley water. Smears showed small and medium sized gram-negative bacilli and diplococci, small gram-positive bacilli and diplococci. No large gram-positive organisms seen. Cultures revealed *Bacillus coli,* aciduric bacilli and cocci. No spore-bearers isolated from raw material or from heated material before or after incubation.

CASE 25.—Q., 7 to 8 months (H. L. H.). Ileocolitis; stools loose. Diet, protein milk and formula. Smears showed small and medium sized gram-negative bacilli, gram-positive diplococci, and large gram-positive bacilli. The latter were abundant but not increased. Cultures revealed *Bacillus coli,* aciduric bacilli and micrococci and terminal-spored bacilli growing in acid broth. No spore-bearers cultivated from raw material or from heated material either before or after incubation.

CASE 26.—S., 9 to 12 months (H. L. H.). Furunculosis, with possible diagnosis of scurvy; stools normal. Diet, protein milk, junket and orange juice. Smears showed small and medium sized gram-negative bacilli, small gram-positive bacilli, gram-positive diplococci and large gram-positive bacilli with occasionally degenerated cytoplasm. Large gram-positive bacilli increased in number in the smears. Cultures revealed *Bacillus coli,* aciduric bacilli, and *Bacillus subtilis.* No increase of spore-bearers on plates from raw or heated material.

CASE 27.—C., 19 months (H. L. H.). Anemia and throat infection; stools normal. Diet, protein milk. Smears showed small and medium sized gram-negative bacilli, small gram-positive bacilli and diplococci and large gram-

positive bacilli much increased in number. The latter were in many instances sporulating with spores in the center and degenerated cytoplasm. Cultures revealed *Bacillus coli*, aciduric bacilli, and *Bacillus subtilis*. No increase of spore-bearers on plates from raw or heated material.

CASE 28.—S., 2 months (H. L. H.). Pyloric stenosis; stools normal. Diet, breast milk and formula. Smears showed small gram-negative bacilli, rather small, long and thin gram-positive bacilli, gram-positive diplococci, and a few large gram-positive bacilli with subterminal spores. Cultures revealed aciduric bacilli and cocci and an unknown aciduric spore-bearer. No other spore-bearers isolated from either the raw or heated material.

THE RELATION OF THE REACTION OF THE URINE TO THE DIET IN INFANTS AND CHILDREN *

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In the treatment of diseases of children there are many indications for changing the reaction of the urine. To bring about this change is often a matter of great difficulty. I believe the majority of failures are due to a lack of a proper dietary regulation, without which drugs may prove of no avail, much to the surprise of the physician.

INDICATIONS FOR CHANGING THE REACTION OF THE URINE

1. The principal indication for changing the reaction of the urine in children is pyelitis, the treatments for which are nearly all founded on these changes. The principles of such treatments are:

(*a*). *Render the urine alkaline,* because it is less irritant to the inflamed mucous membranes and helps to dissolve the mucous and pus.

(*b*) The use of hexamethylenamin, which is considered one of the best antiseptics for the urinary tract. But hexamethylenamin acts only by splitting into ammonia and formaldehyd, the formaldehyd having the antiseptic action; this splitting occurs in appreciable amounts only when the urine is acid.

(*c*). The alternation of these two treatments (*a* and *b*); that is, the changing from alkaline to acid and then from acid to alkaline, etc., hexamethylenamin being given or not during the acid periods.

These changes are supposed to make the surroundings unsuitable for the growth of bacteria, and have the advantage of avoiding the continuous irritative action of the formaldehyd on the urinary tract.

2. *Detroying and preventing crystals and calculi.* The most frequent, and perhaps the only crystals found in children's urine are those formed by uric acid or urates.

(*a*). The urate crystals are formed in acid urines, and some of them, particularly sodium urate, are apt to cause hematuria. An alkaline urine might prevent their formation.

(*b*). *Calculi.* Alkalinity cannot dissolve calculi, but it prevents the precipitation of uric acid and urates, which may occur to a serious extent only in acid urines.

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* Submitted for publication July 20, 1917.

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In a few instances it has been observed that alkalinity caused the breaking up of large stones into small fragments. This cannot be attributed to the solution of the uric acid; the explanation probably is that the calculi were composed of small fragments glued together by mucous, and that the alkalies merely caused the solution of the latter.

3. Acid urine is, as already stated, irritating to the mucous membranes and somewhat to the external genitalia, which often is the cause of masturbation in female children. Acid urine may also possibly cause eneuresis. A change to alkalinity will usually relieve the irritation and dissolve any mucus that may be present.

4. An excess of uric acid in the urine may cause abdominal pains which would be very difficult to diagnose without an examination of the urine. Rendering the urine alkaline might prevent the further formation of the uric acid, or at least make it less troublesome.

5. Alkaline urine has a diuretic action, which, with its antiirritative action, has to be considered in the treatment of nephritis.

6. In cases of acidosis the acid in the urine is a consequence of the excess of acids in the blood. The change of the reaction of the urine is an index of the results of treatment and the progress of the disease.

METHOD FOR TESTING THE REACTION OF THE URINE

The real acidity of the urine, that is, the number of hydrogen ions, can be determined by the indicator method, or by the gas chain method, the former being the simpler.

I used the method of titrating the acidity by means of a tenth-normal or hundredth-normal solution of sodium hydrate which is added up to neutralization, phenolphthalein being used as the indicator. This is a very simple method and seems to have been sufficiently accurate as far as the final results are concerned.

If one desires to compute the number of grams of hydrogen in each 100 c.c. of urine it is necessary to multiply by 0.1 the number of cubic centimeters of tenth-normal sodium hydrate solution used in neutralizing the 10 c.c. of urine.

Dry potassium oxalate must be previously added to the urine in order to precipitate the lime salts which otherwise would precipitate the phosphates when the hydrate is added. Six grams should be added for each 10 c.c. of urine used.

When litmus is used, it must be remembered that the urine may be found alkaline, while it may be acid to phenolphthalein.

In testing the degree of alkalinity of a urine, I used a sulphuric acid solution equivalent to the strength of the sodium hydrate, with the same indicator.

HOW TO CHANGE THE REACTION BY DIETARY PROCEDURE

In studying the relation of diet to urinary reaction, I have considered the following phenomena, which, in part, were responsible for this investigation:

1. The urine of the carnivora is acid, and that of the herbivora is alkaline.

2. The acidity of the urine of the carnivora depends especially on the products of protein metabolism. Sulphur occurs in an unoxidized condition. Phosphoric acid occurs in nucleic acid, in neucleoproteins, in pseudonucleins and in lecithin. During metabolism these are oxidized into sulphuric acid or set free as phosphoric acid, thus accounting for the acidity of the urine.

3. There are also organic acids such as phenylacetic, benzoic, uric, urocyanic, etc., which are formed by oxidation of certain protein constituents, and contribute also to the acidity of the urine.

Nitrogen from the proteins is eliminated in the unoxidized form as urea for the most part, but this is a very weak base, practically neutral in reaction, and hence is not capable of neutralizing the acid produced.

4. The alkaline reaction of the urine of the herbivorous animals is due to the fact that vegetables and fruits contain salts. Some of these salts are neutral; others are acid salts of dibasic or polybasic acids or other carboxylic acids, as, for example, acid potassium malate, citrate, acetate, tartrate, etc.

On oxidation in the body these salts are burned to carbonates. Some of them are excreted through the lungs in the form of carbonic acid, leaving the disassociated base — usually sodium or potassium — to reunite with the very weak acid, carbonic acid, and in this form is excreted in the urine. These carbonates have an alkaline reaction owing to their giving rise to free alkali by hydrolysis. This explains why lemonade or orange juice makes the urine alkaline.

The vegetable proteins, on the other hand, are not absorbed as well as the animal proteins, and their products of metabolism are more apt to be found in the stools.

5. In starvation, the acidity of the urine increases, owing to the breaking down and digestion of the tissue proteins, which of course are animal in origin.

6. During the infectious diseases, also, the absorption of protein products is increased, especially those of the nucleic group, giving rise to an especially acid urine.

From all these facts, I arrive at two theoretical conclusions:

1. The more protein in the food (especially of the nucleic type) the more acid the urine.

2. A diet of vegetables, especially of those poor in proteins, diminishes the acidity of the urine.

With these theories as a starting point, the following experiments were undertaken, first on babies, and then on older children. Bottle fed babies rather than breast fed babies were used because of the impracticability of changing the percentage of the elements of the breast milk. This was readily done with the bottle feedings. The three babies used were on formulas of which the exact percentages of fat, carbohydrate and protein were known. These percentages were changed at will during the course of the experiments.

The results are shown in Charts 1, 2 and 3. The circles represent the grams of protein in the diet per kilo of body weight; the solid dots represent the number of cubic centimeters of tenth-normal sodium hydrate or sulphuric acid solution necessary to neutralize 10 c.c. of the whole twenty-four-hour amount of urine.

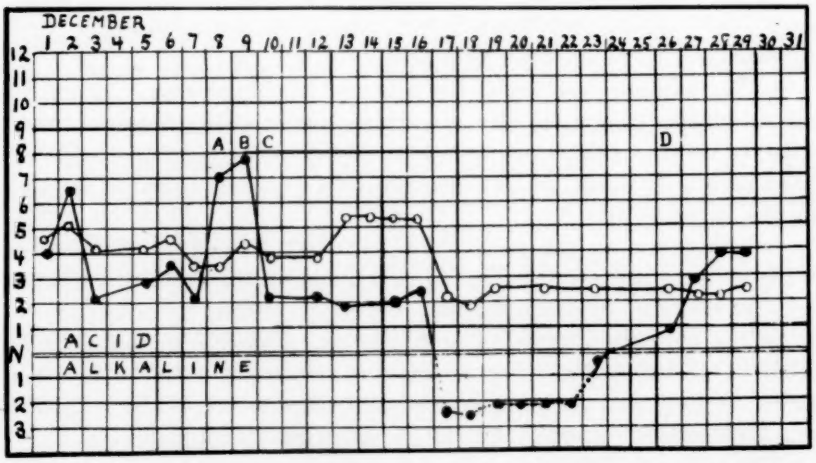

Chart 1.—The relation of feeding and other factors on the reaction of the urine in an infant: *A*, infant lost 100 gm.; *B*, lost 150 gm.; *C*, regained 100 gm.; otitis media.

EXPERIMENTS

Baby 5 months old (Chart 1). Started on a formula containing fat, 2 per cent.; dextrimaltose, 6 per cent.; protein, 2.50 per cent. (880 c.c.). As the baby weighed 4,620 gm., it was getting 4.7 gm. of protein per kilo of body weight. The acidity of the urine on the first day was 4; that is, it took 4 c.c. of tenth-normal sodium hydrate solution to neutralize 10 c.c. of urine. The next few days, although the percentage of protein in the food was not changed, the baby's daily total food intake and weight necessarily varied a little, thus accounting for the irregularity in the curve representing the relation of grams of protein in the food to the kilo of body weight. This variation holds throughout all these experiments.

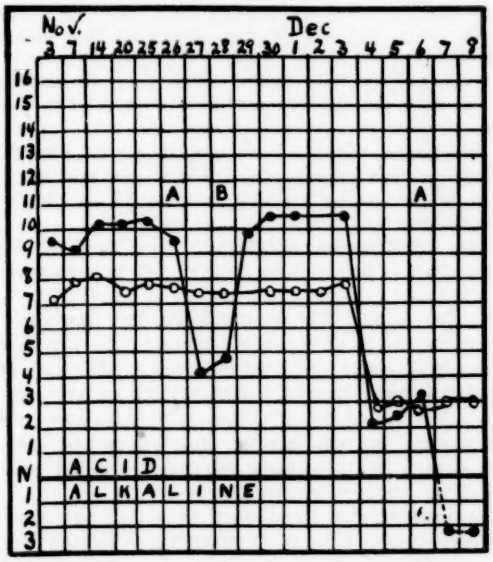

Chart 2.—The relation of feeding and other factors on the reaction of the urine in an infant: *A*, infant fed cereal soup; *B*, cereal soup omitted.

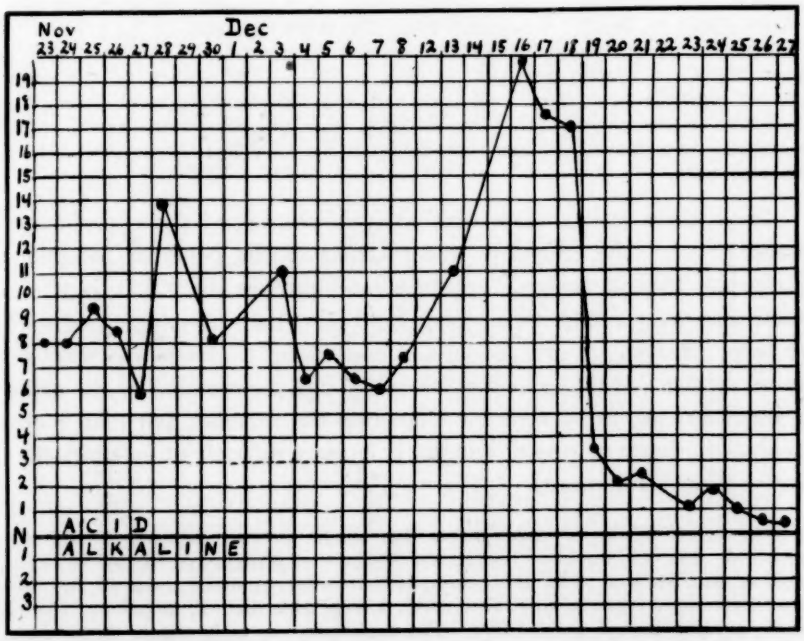

Chart 3.—Irregular acidity of the urine on an unchanged diet.

December 8, the acidity suddenly rose to 7 c.c. with no change in the food. It happened that the baby lost 110 gm. of weight on that day, due to a nasopharyngitis accompanied by a slight fever. December 9, there was a further loss of 150 gm.; the acidity rose again. December 10, the baby was better and gained back 100 gm.; the acidity immediately dropped to the level at which it was before the onset of the fever and remained there.

December 17, the protein in the food was dropped from 2.50 per cent. to 0.90 per cent. This protein represented 1.9 gm. per kilo of body weight on December 18. The urine became alkaline, so that 2.2 c.c. of tenth-normal sulphuric acid was necessary to render 10 c.c. of it neutral.

December 26, an otitis media developed. The acidity of the urine rose immediately. A transfer of the baby to another bed made further experimentation impossible.

Baby 8 months old (Chart 2), weight 5,110 gm., November 3 was given a formula representing fat 2.50 per cent., lactose 5.50 per cent., protein 3.20 per cent. The baby took about 1,170 c.c. of food a day; this afforded 7.3 gm. of protein per kilo of body weight. The acidity of the urine remained at about 9 or 10 c.c. until November 27, when a sharp drop in the acidity was noted. I learned that on the day before, cereal soup had been ordered twice a day. Two days later I asked to have the cereal omitted, and without it the acidity again rose to its former level.

December 3, the protein was diminished from 3.20 per cent. to 2 per cent. This new formula gave about 2.9 gm. of protein per kilo of body weight. The acidity decreased immediately to 2 or 3 c.c., but the urine did not become alkaline. As a matter of interest, the cereal soup was started again December 6, and a marked degree of alkalinity resulted—over 2 c.c.

Baby 8 months of age (Chart 3), was started, November 23, on a formula containing 3 per cent. protein, representing 6.6 gm. of protein per kilo of body weight. It is to be noticed how irregular the acidity was in this urine, although the food was unchanged. The acidity began at 8 c.c. and oscillated widely, rising as high as 20 c.c. I cannot explain this wide variation.

December 18, the protein was reduced from 3 per cent. to 1 per cent., giving about 2 gm. of protein per kilo of body weight. The acidity of the urine dropped immediately to 2 c.c. and remained at about the same level during the remainder of the experiment. The addition of a vegetable soup would in all probability have turned the urine alkaline, but the baby left the hospital before this was tried.

These three charts show well what it was hoped the experiments would prove — the effects on the reaction of the urine of infection, starvation, loss of weight, vegetable and protein diets. It is to be noted that the proteins were never fed below the physiologic needs of the body; that is, 1.5 gm. per kilo of body weight. Emphasis must be placed on the fact that it is possible to change the reaction of the urine in bottle fed babies by a simple change in the formula, but that in the presence of fever or loss of weight, it is more difficult to make it alkaline by these same means.

The second series of experiments was made on two children, 5 and 5½ years old. In their diets, of course, the amount of proteins could not be regulated as accurately as with the babies, but in order to make them as accurate as possible, four types of diet were used: 1, vegetable diet; 2, mixed or "House Diet"; 3, milk diet; 4, animal diet. The accompanying table shows the average food percentage and calories of each diet.

TABLE SHOWING FOOD PERCENTAGE AND CALORIES OF VARIOUS DIETS

I. VEGETABLE DIET *

Breakfast	Amount, Gm. or C.c.	Fats, Gm.	Carbohyd., Gm.	Proteins, Gm.
Cereal (rice)	100	1.50	2.00	8.00
Bread	15	0.50	15.00	3.00
Butter	5	5.00		
Dinner				
Bread	30	1.00	30.00	6.00
Tomato	30	0.07	1.40	0.42
1 potato.....	...	1.00	20.00	1.70
Asparagus	30	0.30	0.80	0.45
Cereal	100	1.50	2.00	8.00
Butter	...	5.00		
Supper				
Toast	15	0.50	15.00	3.00
Peas	40	0.40	1.20	8.00
Orangeade	120	0.25	21.25	1.50
Apple sauce.....	125	1.00	46.50	0.25
Total		18.02	155.15	40.32

Total calories, 1,008.03

*3 gm. protein per kilo; 80 calories per kilo.

II. MIXED DIET †

Breakfast				
Potato	...	1.00	20.00	1.70
Milk	120	4.80	5.40	3.80
Bread	30	0.50	15.00	3.00
Dinner				
Chicken	45	4.50	.....	10.00
Soup	100	....	0.60	3.48
Bread	30	0.50	15.00	3.00
Butter	...	5.00		
Potato	...	1.00	20.00	1.70
Supper				
Milk	200	8.00	9.00	6.40
		1.20	1.60	6.40
		0.50	15.00	3.00
Total		27.00	101.60	42.48

Total calories, 913.05

†72 calories per kilo; 3.5 gm. protein per kilo.

III. MILK DIET ‡

Milk	1,000	40.00	45.00	32.00

Total calories, 687.8

‡2.56 gm. of protein per kilo; 55 calories per kilo.

IV. ANIMAL DIET §

Breakfast				
1 egg.....	...	5.00	....	7.00
Milk	180	7.20	8.10	5.76
Bread	15	0.25	8.00	1.50
Dinner				
1 egg.....	...	5.00	....	7.00
Milk	210	8.45	9.45	6.72
Broth	90	0.50	....	10.00
1 chop.....	60	6.00	....	14.00
Supper				
Milk	180	7.20	8.10	5.46
Bread	15	1.50	10.00	1.50
1 egg.....	...	5.00	.....	7.00
Total		46.10	43.65	65.94

Total calories, 880.55

§5 gm. protein per kilo; 70 calories per kilo.

The weight of the two children was about the same (12,400 and 12,550 gm.); therefore, they were getting about the same number of calories and the same amount of protein per kilo. The proportions were as follows:

	Calories Per Kilo	Proteins Per Kilo, Gm.
I. Vegetable Diet.....	80	3.0
II. House Diet.....	72	3.5
III. Milk Diet.....	55	2.6
IV. Animal Diet.....	70	5.0

The number of calories per kilo changed very little with the different diets excepting with the milk diet, because the children never took the amount required. The vegetable diet gave the greatest number of calories and the animal diet the greatest amount of protein per kilo.

As what I desired to determine in these experiments was the acid concentration of the urine, as well as the total acidity in twenty-four

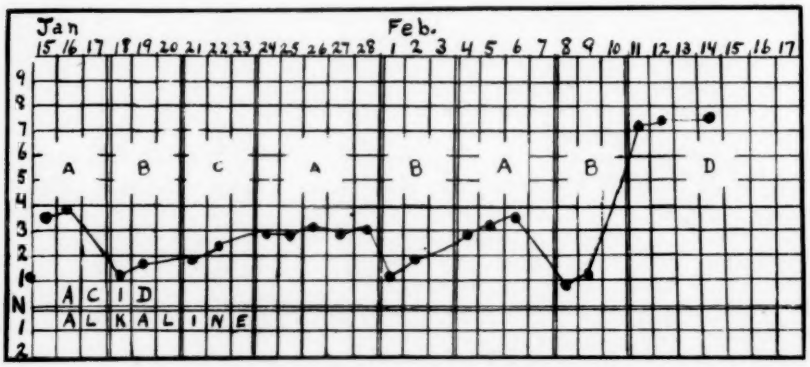

Chart 4.—Influence of diet on the reaction of the urine in a child aged 5½ years: *A*, house diet; *B*, vegetable diet; *C*, milk diet; *D*, animal diet.

hours, especial care was necessary in giving the children the same amount of water every day, because it is evident that with the same diet the total acid eliminated in twenty-four hours ought to be about the same; but the concentration of the same quantity of urine taken from the whole twenty-four-hour amount would vary with the total volume eliminated; in other words, with the amount of water ingested.

The curves in Charts 4 and 5 show the changes in the acidity of the urine in the two children on the four diets given. The acidity represented by the dots denotes the number of cubic centimeters of tenth-normal solution of sodium hydrate required to neutralize 10 c.c. of urine taken from the total twenty-four-hour amount.

The changes in the diets were made twenty-four hours before testing the urine.

The curves do not show as wide changes as in the babies, because the changes in the amount of protein in the diet could not be regulated as well. While in the babies the protein changed from 2 to 8 gm. per kilo, in these children they could be changed only from 2.6 to 5 gm. per kilo of body weight.

The two curves (Charts 4 and 5) are remarkable for their similarity, although with some minor differences. The first decrease after changing from the "house diet" to the vegetable diet, is wider in Curve 4 than in 5; and, on the other hand, the small increase after changing from the vegetable to the milk diet is more marked in Curve 5 than in 4. The noteworthy points in these curves are:

1. The acidity of the urine decreased on the vegetable diet and rose more and more on adding food of animal origin. This change is very noticeable in both curves between February 9 and 11, when the vegetable diet was changed to an animal diet.

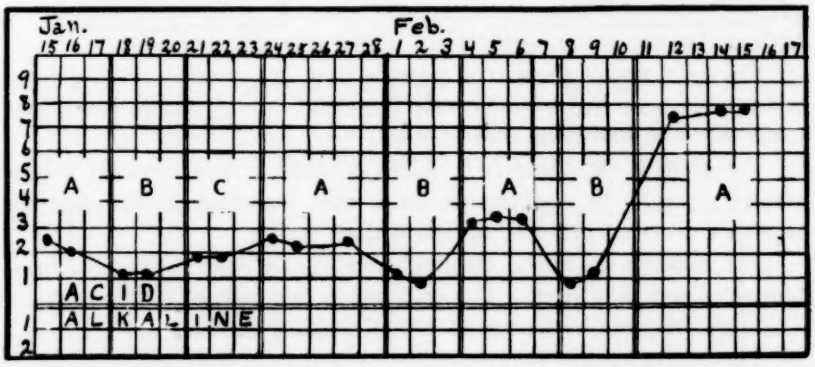

Chart 5.—Influence of diet on the reaction of the urine in a child aged 5 years: *A*, house diet; *B*, vegetable diet; *C*, milk diet; *D*, animal diet.

2. The difference in effect of the vegetable diet and milk diet was practically none. Theoretically, the acidity ought to increase with the milk diet (although not as much as with the animal diet), but the children only took on an average of 1,000 c.c. of milk daily, which only represented 2.6 gm. of protein per kilogram, being the smallest amount of protein taken during the experiment.

THE USE OF DRUGS

Only a superficial study was made of the drugs generally used to change the reaction of the urine, as it requires a great deal of time and hardly comes within the scope of this paper.

For making the urine acid, either acid sodium phosphate or sodium benzoate is generally used. The former is eliminated in its acid form

and the latter combines in the body with glycocoll, forming hippuric acid, which is excreted in the urine.

The curves representing the acidity and alkalinity of the urine as the result of the administration of drugs, do not represent the acidity or alkalinity of a fixed volume, but the acidity or alkalinity of each micturition, because in these experiments it was intended to study, not the daily changes, but rather the changes at frequent intervals during the twenty-four hours, on account of the fleeting action of the drugs.

Curve 6 shows that 2 grains (0.13 gm.) of acid sodium phosphate made the acidity increase to 12.5 c.c. It then decreased immediately after stopping the drug, reaching nearly the initial point (5.2 c.c.) eight hours after the last dose.

Curve 7 shows that the acidity remained the same for two hours after the first dose of 2 grains of sodium phosphate, which was probably due to a slower absorption, as three hours after the second dose the acidity rose from 2 c.c. to 14 c.c. As with the acid sodium phosphate, it dropped after the drug was stopped and reached 1.6 c.c. (a little under the initial point) eight hours after the last dose.

Curves 8 and 9 show that a single dose of 5 grains (0.33 gm.) of sodium benzoate had a similar effect, which began to diminish six hours after the dose had been administered.

From these experiments it seems conclusive that the acid sodium phosphate and the sodium benzoate must be given in frequent and repeated doses in order to maintain their action.

For making the urine alkaline potassium citrate or sodium bicarbonate is generally used. The indications for making a urine alkaline are more frequent in therapeutics than making it acid.

The following are two experiments which I think are worth mentioning:

One dose of 20 grains (1.25 gm.) of potassium citrate was given to a 5-year-old child with an ordinary "House Diet" to which about 15 gm. of extra meat were added. Then the same dose of the same drug was given while the child was taking a vegetable diet, adding as an extra, about 100 c.c. of orange juice (see Charts 10 and 11).

The results were that in the first part of the experiment the urine was acid and turned alkaline when the diet was changed.

The same experiment was made with the same diets, but giving 40 grains of sodium bicarbonate instead of the potassium citrate, and the same results were obtained (see Charts 12 and 13).

These last experiments are alike in results to those of the babies and demonstrate clearly the importance of the diet when it comes to changing the reaction of the urine.

Very often the potassium citrate and the sodium bicarbonate cause, especially in babies, digestive disturbances because of their action on the stomach. Such is the case in pyelitis and cystitis when one is

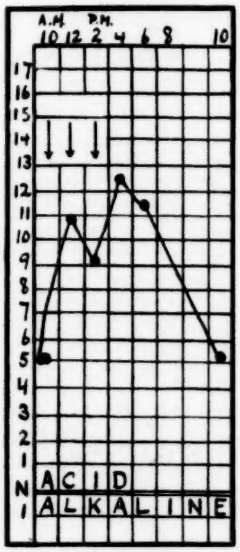

Chart 6

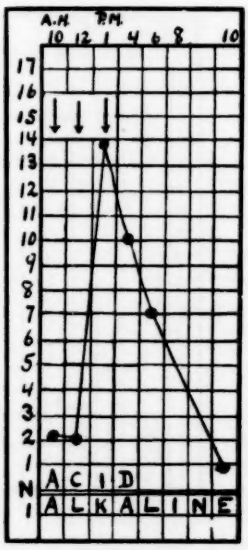

Chart 7

Chart 6.—Action of sodium acid phosphate in a child aged 5½ years, on a house diet. Arrows point to administration of 2 grains of the drug.

Chart 7.—Action of sodium acid phosphate in a child aged 5 years, on a house diet. Arrows point to administration of 2 grains of the drug.

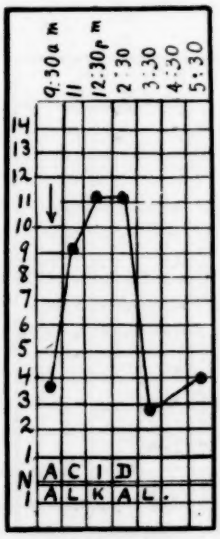

Chart 8

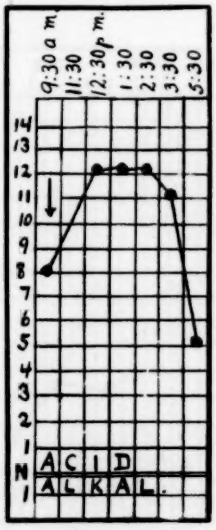

Chart 9

Chart 8.—Action of sodium benzoate in a child aged 5 years, on a house diet. Arrow points to administration of 5 grains of the drug.

Chart 9.—Action of sodium benzoate in a child aged 5½ years, on a house diet. Arrow points to administration of 5 grains of the drug.

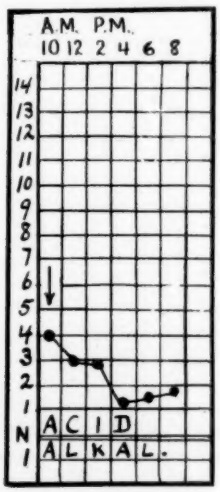

Chart 10

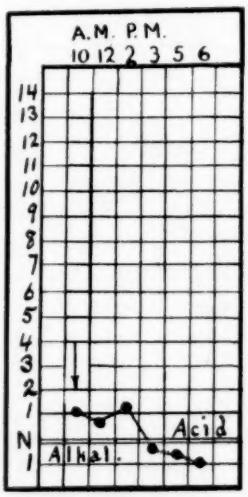

Chart 11

Chart 10.—Action of potassium citrate in a child aged 5 years, on a house diet. Arrow points to administration of 20 grains of the drug.

Chart 11.—Action of potassium citrate in the same child as in Chart 10, on a vegetable diet. Arrow points to administration of 20 grains of the drug.

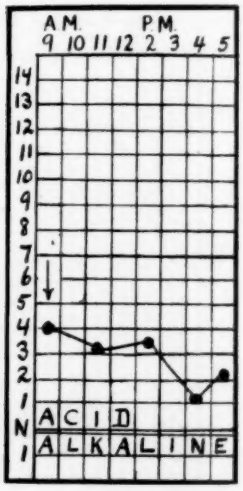

Chart 12

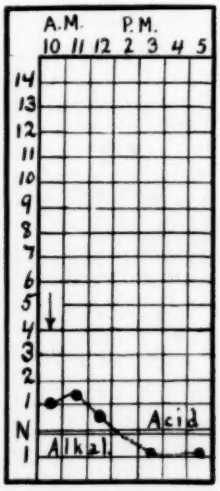

Chart 13

Chart 12.—Action of sodium bicarbonate in a child on a house diet. Arrow points to administration of 40 grains of the drug.

Chart 13.—Action of sodium bicarbonate in the same child as in Chart 12, on a vegetable diet. Arrow points to administration of 40 grains of the drug.

obliged to give very large doses of the drug. Hole has said: "The citrate of potash in this condition [pyelitis] is apt to cause diarrhea or vomiting. It is therefore wise to give no more than 5 or 10 grains."

The two following cases will illustrate this a little better:

S. M., 2½ months old, in the Massachusetts General Hospital with acute pyelitis, was fed on modified milk and a small amount of breast milk. He was treated in the beginning with hexamethylenamin, but on account of a suspicion of nephritis the treatment was changed to rendering the urine alkaline. For this purpose he was given 15 grains of potassium citrate every four hours.

After three days of this new treatment I examined the urine before the morning dose of the medicine was given, the acidity of which was 4.2 c.c.; in the evening, two hours after the third dose, the acidity was 1.2 c.c. By that time the baby had taken a little more than 1.11 gm. of potassium citrate per kilo, which is a larger dose than is ordinarily given, even when a large dose is intended.

The baby was getting in his food, 4.27 gm. of protein per kilo. Perhaps if the protein had been diminished the urine would have become alkaline, but I did not have the opportunity to follow up this case.

M. M., 2 years old, having pneumonia and laryngitis, following the removal of a foreign body from a bronchus, had pyelitis. In order to render the urine alkaline, she was given 30 grains of potassium citrate every 2½ hours.

The urine was examined in the morning, before the first dose, and the acidity was found to be 6.6 c.c., and after taking 120 grains of the potassium citrate it was still acid (4.3 c.c.).

It is evident from these cases how difficult it is to turn the urine alkaline, when there is an infection in the urinary tract, without regulating the diet according to the schemes described.

CONCLUSIONS

1. The difficulties which one often encounters in changing the reaction of the urine in children are very much diminished when a suitable diet is prescribed.

2. Diminishing the amount of protein in the diet to 2 gm. and sometimes to 3 gm. per kilo will often in itself render the urine alkaline without using a drug.

3. In cases in which the amount of protein reaches the lowest physiologic limit without turning the urine alkaline, one may continue to diminish the acidity by adding vegetables to the diet.

4. In older children with whom the food is more varied, one can do much by regulating the diet in accordance with the principles outlined in this article.

5. In cases in which vegetables are used to turn the urine alkaline, one must use, if possible, vegetables in which the amount of protein is smaller in proportion to the salts, like fruits, rice, wheat, etc., rather than those which have a large amount of protein like pease, beans, etc. Vegetables which contain purins, like coffee, tea and cocoa, may increase the acidity, because they may produce uric acid.

6. In cases of infection, especially in infections of the urinary tract, and in cases of starvation, the urine tends to become more acid than in other conditions; therefore it is more difficult to render it alkaline.

7. When drugs are used without the proper diet, the doses necessary to make the urine alkaline are much larger than is ordinarily believed.

8. The action of drugs (either acid or alkaline) is very fleeting; therefore they should be given in large and frequent doses.

I desire to express my gratitude and thanks to Drs. Fritz B. Talbot and Joseph I. Grover for the suggestions and assistance and for facilities provided for my experiments, the former physician in the Massachusetts General Hospital and the latter in the Infants' Hospital.

THE BANANA AS A FOOD FOR CHILDREN *

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AND
ANTON R. ROSE, M.S., PH.D.
NEW YORK

The banana has been regarded as of doubtful value in the dietary of children, and it seemed worth while to investigate the merits of a food so abundant, economical and popular, and to determine the best ways of using it to get full nutritive value without digestive disturbances.

The chief food constituents of the banana are the carbohydrates which amount to about 22 per cent. In the green fruit these are in the form of a starch, but on maturing this is converted first into sucrose and finally into invert sugar. The proteins amount to less than 1 per cent. and their nature is at present unknown. The fat and fiber are negligible. The ash is important both in amount and kind of its constituents. Fruits are generally recommended for their vitamin content, and it is reasonable to think that the banana may have its share. These dietary accessories or vitamins are at present being investigated. The main value of the fruit lies, then, in its carbohydrates, ash, and possibly its accessories. The composition of the banana is compared with that of some other common fruits and the potato in Tables 1 and 2. The figures in these tables refer to the edible portion of the fresh food.

While theoretical considerations and practical tests made in this institution show that the banana is a valuable food, it is not to be regarded as a complete food in itself or to be recommended as the sole or main part of a child's diet. It cannot take the place of such staples as milk, cereals, or potatoes, though it may in part replace the latter two at times. When *fully ripe* it is a good fruit for children and may be given freely without danger or harm except as it may crowd out other foods more essential for growth. *The underripe uncooked fruit should never be given to children. Bananas that are merely yellow are not ripe.* Characteristic changes take place in the peel as the fruit matures. The thick, turgid covering of the green banana becomes thinner and more pliable, the color passes from green

* Submitted for publication July 24, 1917.

* From the Department of Pediatrics and the Laboratory of Pathological Chemistry, New York Post Graduate Medical School and Hospital.

TABLE 1.—Comparative Composition of Some Common Fresh Fruits and Potatoes *

Fruit	Protein	Carbohydrate	Calories per Pound	Grams Required to Yield 100 Calories
Banana	1.3	22.0	447	101
Grapes	1 3	19.2	427	104
Plums	1.0	20.1	383	118
Cherries	1.0	16.7	354	128
Raspberries	1.7	12.7	290	150
Pears	0.6	14.1	288	158
Apples	0.4	14.2	285	159
Blackberries	1.3	10.9	262	173
Apricots	1.0	13.4	247	172
Orange	0.8	11.6	233	195
Peaches	0.7	9.4	187	242
Pineapples	0.4	9.7	197	233
Lemons	1.0	8.5	201	226
Cantaloup	0.8	9.3	180	252
Strawberries	1.0	7.4	177	269
Watermelons	0.4	6.7	136	332
White potatoes	2.2	18.4	378	120
Sweet potatoes	1.8	27.4	558	81

* Olives, sweet potatoes, and corn are the only common fresh fruits and vegetables that have a higher fuel value than the banana. Other fruits and vegetables that may be classed with the banana as having approximately 1 calory per gram are grapes, plums, and potatoes as given above, together with figs, which are almost identical with bananas, and dates with a fuel value equal to that of the grapes. This table is compiled from data taken from Rose, M. D. S.: Laboratory Handbook for Dieteties, New York, and was used in part in a former paper: Myers and Rose: Jour. Am. Med. Assn., 1917, **68**, 1022.

TABLE 2.—Ash Constituents of Some Common Fruits *

Fruit	Ash Constituents of Fruits in Percentage of the Edible Portion							
	CaO	MgO	K_2O	Na_2O	P_2O_5	Cl	S	Fe
Bananas	0.01	0.04	0.50	0.02	0.055	0.20	0.013	0.0006
Grapes	0.024	0.014	0.25	0.03	0.12	0.01	0.024	0.0013
Plums	0.025	0.02	0.25	0.03	0.055	0.01	.....	0.0005
Cherries	0.03	0.027	0.26	0.03	0.07	0.01	.....	0.0005
Pears	0.021	0.019	0.16	0.03	0.06	....	.....	0.0003
Apples	0.014	0.014	0.15	0.02	0.03	0.004	0.005	0.0003
Oranges	0.06	0.02	0.22	0.01	0.05	0.01	0.013	0.0003
Peaches	0.01	0.02	0.25	0.02	0.047	0.01	0.01	0.0003
Lemons	0.05	0.01	0.21	0.01	0.02	0.01	0.012	0.0006
Muskmelons	0.024	0.02	0.283	0.082	0.035	0.041	0.014	0.0003
Strawberries	0.05	0.03	0.18	0.07	0.064	0.01	.....	0.0009
Watermelons	0.02	0.02	0.09	0.01	0.02	0.01		
White potatoes	0.016	0.036	0.53	0.025	0.14	0.03	0.03	0.0013
Sweet potatoes	0.025	0.02	0.47	0.06	0.09	0.12	.....	0.0006

* Sherman, H. C.: Chemistry of Food and Nutrition, p. 332.

to pale yellow, deepening to golden yellow with brown spots, and finally becoming brown all over. Bananas are edible raw when the brown spots appear and need not be refused as overripe when the peel is entirely brown if the covering is intact and the pulp shows no signs of fermentation. The brown color of the ripening must not be confused with the brown patches due to bruising (Table 3).

TABLE 3.—CHANGES TAKING PLACE IN THE BANANA DURING THE PROCESS OF RIPENING *

Days off Boat	Per Cent. of Peel	Reducing Sugar in Terms of Glucose, per Cent.	Sucrose in Terms of Glucose, per Cent.	Total Sugar in Terms of Glucose, per Cent.	Remarks on the Condition of the Peel and Pulp
2	40	0.6	1.2	1.75	These bananas were very green and firm; could be handled like cordwood without harm to fruit; the peel adheres to pulp; total carbohydrates about 22 per cent.
4	39	1.2	4.6	5.8	Greenish yellow with pea green edges; peel still adheres to pulp; taste bitter and astringent; starch about 15.5 per cent.; may be baked at this stage, but preferable to hold until two days later
6	36	2.9	12.5	15.4	Yellow except at extreme edges and tip; there is a beginning of the golden tints; the peel does not adhere to the pulp; the bitter taste has largely disappeared; starch about 7.5 per cent.; very good for baking
8	32	4.1	11.9	16.0	Greenish cast to edges; otherwise a full yellow; starch 5 per cent.; flavor somewhat herbaceous; this fruit should not be eaten raw though it is frequently consumed at this stage
12	26	5.4	11.7	17.1	Golden yellow with some suggestion of green on edges and tip; few brown specks; herbaceous flavor; this fruit may be eaten raw by adults but it is preferable to hold it over one more day before serving to children
14	26	6.9	11.7	18.5	Full golden yellow with many small brown spots; starch 3 per cent.; flavor delightful; perfectly safe to consume this fruit in any reasonable quantities
15	26	7.1	11.1	18.8	Extensive patches of light brown; starch less than 1 per cent.; excellent flavor and mellow texture; at optimum condition for eating raw

* The above figures and remarks are based on a bunch of bananas received from a refrigerant boat in November. The course of the ripening process is influenced by the temperature, and different data would be recorded from a bunch examined in the summer months. In warm weather the pulp advances more rapidly than the peel and a banana in a golden yellow skin may be as mellow as the light brown fruit.

To establish clearly the true nutritive position of the banana, a large number of carefully controlled feeding experiments have been conducted on normal adults, invalids and children, including infants. The fruit has been used in various degrees of ripeness. It was impossible to feed the raw banana in the early stages of yellowing on

account of the bitter astringent still present. Fruit of this ripeness, however, served to adults and children, baked in the skin as in baked potatoes, and mashed with butter before eating, proved quite acceptable.[1] The underripe banana when baked is utilized to about the same extent as the potato and is similar to this vegetable in its nutritive value. There was in no case any suggestion of deleterious effects from eating the banana baked or raw when fully ripe.

In the late yellow stage, before the development of the distinct golden color, the raw fruit can be consumed by normal individuals in small quantities (say one or two bananas) without discomfort or apparent harm. Nevertheless the authors are of the opinion that the raw fruit should not be given to children at this stage. In one instance, where the plan was to have the banana at this stage of ripeness as the sole food for three days, a distinct disturbance developed after the fifth meal, manifesting itself in nausea and vomiting. It should be added, however, that 1.5 kg. had been taken without any uncomfortable feelings whatever before the symptoms manifested themselves. No such symptoms have been produced by the fruit in the golden yellow stage of ripeness. Several adults were kept on a diet consisting almost exclusively of milk and fully ripe bananas for periods of a week. For example, in one case the diet was composed of 1,000 gm. banana, 1 quart of milk, and 1 cup sweetened coffee per day for one week. The experience was one of comfort, satisfaction, and unimpaired efficiency, and the diet was more pleasurable than that of a similar period in which the banana was replaced by its calorific equivalent of fresh white wheat bread. The utilization of the banana carbohydrate in these cases was 98.8 per cent.

The experiments on children consisted in feeding carefully controlled dietaries planned to be adequate and normal in every way. These were divided into periods of from three to seven days. "Control" periods in which no banana was served were either made up of the regular ward diet or of a similar vegetarian diet. In the latter case the same diet was repeated each day of the period. Very underripe bananas were excluded, as these are known to be unsuitable. In the "banana test" period the banana was introduced as a substitute for the more starchy foods, such as cereals and potato, and ranged in amount from one small banana to three-quarters of the day's carbohydrate quota. With one exception the banana was mashed to prevent the child from bolting it in unmasticated lumps. The food served was weighed on a Harvard trip scales to within 1 gm. and when it was not

1. The banana may be baked at all stages, even when very green, but the pulp of the banana baked before the peel has assumed the yellow color becomes tough and dry. The green banana is desiccated and ground into a meal in which form it is sometimes used.

all eaten the returned parts were also weighed and recorded. The stools were collected quantitatively and analyzed. The reaction to litmus and reduction of picric acid were employed to detect any signs of intestinal fermentation. The determinations of carbohydrates were made by the modified picric acid method.[2] The reduction of picric acid after acid hydrolysis, expressed in terms of glucose, is assumed to represent the starch and dextrin of the stools. The total carbohydrate thus determined in the stools subtracted from the ingested carbohydrates gives the apparent utilization of this food element and is expressed in terms of per cent. of the carbohydrate consumed. The foods used were not themselves analyzed but calculated from tables used in planning the diets.[3]

The nurses in charge of the children were interested in the experiments and gave a hearty cooperation in the work. The probable errors, therefore, were reduced to two; that of obtaining food surreptitiously, which would weight the balance against the banana, and secondly the loss of stools, which would weight the balance in favor of the banana; but, on the other hand, loss of stools would be certain of detection by both the magnitude of the deflection from the mean daily carbohydrate elimination and the conflict with the recorded defecation habit. Most of the known and suspected mishaps have been ruled out by eliminating the cases from further consideration or in the cases in which they have been included in this study by omitting the defective days from the calculations.

DATA FROM THE EXPERIMENTS

The reliable data from these experiments are briefly reviewed in the following:

CASE 1.—A. R., aged 5 years, girl, weighing 35.6 pounds. Rheumatic endocarditis; constipated, requiring enema and purgatives before and after the test, also during the test. The diet before the test was described as "soft." There were two control periods consisting of farina, bread, butter, oatmeal, potatoes, soup, milk, and cocoa. In two other periods the potato and part of the cereal were replaced by bananas, mashed in the one period and thickly sliced in the other. The banana was partly golden yellow, but not yet darkened or spotted with brown specks. There were, therefore, unaltered starch grains left in the pulp. The subject took the meals as well as could be expected and no mishaps occurred except on the last day when a distaste for the foods developed and the noon meal was regurgitated. This day is not included in the numerical data of Tables 4 and 5.

CASE 2.—F. C., aged 6 years, boy. Congenital malformation; mentally subnormal; parentage and history unknown; laboratory findings and observations indicate physiologic normality. This subject was on the test at the same time as Case 1 and the same diet and management of details were employed. No vitiating factors are known. These two cases were the first cases used in

2. Myers, V. C., and Bailey, C. V.: Jour. Biol. Chem., 1916, **24**, 147.

3. Rose, M. D. S.: Laboratory Handbook for Dietetics, New York, 1912; and Feeding the Family, New York, 1916.

the baby ward for this study and were very closely watched by all concerned. The numerical data relating to them are tabulated in detail as illustrative of the whole plan and the following cases are presented more briefly (see Table 4).

TABLE 4.—NUMERICAL DATA PERTAINING TO CASES 1 AND 2

	Case 1				Case 2		
	Control Period May 4-7	Banana Period May 8-11	Control Period May 12-15	Banana Period May 16-18	Control Period May 4-7	Banana Period May 8-11	Control Period May 12-15
Carbohydrate intake, gm. .	210	320	210	320	210	320	210
Amount of feces, gm.	221	306	260	247	228	200	173
Reducing substances in the feces after acid hydrolysis in terms of glucose, per cent.	1.44	2.8	1.2	2.83	1.8	2.5	1.5
Estimated amount of carbohydrate in the feces in terms of glucose, gm. ..	3.1	8.6	3.2	7.0	4.1	5.6	2.6
The apparent utilization of the carbohydrates in per cent. of the carbohydrates ingested	98.5	97.3	98.7	96.8	98.1	98.3	98.8

CASE 3.—R. P., aged 3 years, girl, weighing 21.6 pounds. Congenital syphilis. The Wassermann test was negative. The stools were mostly normal and regular. During the stay in the hospital the bowels failed to move seven days, three of which were due to an attack of constipation prior to the test and three to an attack during the test. The appetite was good and all meals were taken regularly throughout. The control period of five days was based on a daily diet consisting of farina 80 gm., bread and butter 120 gm., potatoes 60 gm., rice pudding 100 gm., 1 egg, 1 baked apple, 100 c.c. soup and 400 c.c. milk. The banana test diet was the same, with the substitution of ripe bananas for 60 gm. of the cereal, and all of the rice, potatoes, and apples in calorifically equivalent amounts. The carbohydrate in the stool varied very little from day to day and for the control period averaged 0.71 gm. and for the banana days, 0.77, indicating practically complete utilization.

CASE 4.—R. S., aged 5½ years, girl weighing 40.56 pounds. Convalescent from typhoid fever; constipated; required frequent enema; vomited; abdominal pains both before and during the experiment. The diet used was the same as that in Case 3. Several stools were lost. For those days of the test when the control of the experiment was reasonably good the average of the carbohydrate in the stools was 0.11 gm. for the banana days and 0.15 and 1.2 gm. for two of the control days. The unusually low elimination of carbohydrate corresponds to days of unassisted defecation, yielding very firm stools; the higher figure represents three days of enema treatment.

CASE 5.—D. C., aged 6½ years; boy weighing 33 pounds. Tuberculosis of the ankle; constipated, but the stools were regular during the experiment. This child's appetite was subnormal. In the control period the regular ward diet was employed, the following being a sample day: cereal 140 gm., milk 550 c.c., meat 10 gm., potatoes 40 gm., apple sauce 30 gm., and broth 150 c.c. There were two periods in which banana replaced part of this regular diet; in the one 200 gm. of banana were prescribed and in the other 400 gm., the other food being left to the choice of the patient and the nurse; but the quantities actually consumed were weighed and recorded. An insufficient amount of food was taken in all of the periods, the lowest being in the control period

in which only 800 calories or about one-half of the normal amount was taken. In the banana periods the calories were brought up to 1,000. The amounts of carbohydrate in the stools of the control period were unusually uniform from day to day, varying less than 0.4 gm., with an average for the period of 2.50 gm. In the 200 gm. banana period the carbohydrates in the stools averaged 2.93 gm., or a trifle more than that of the control period, but more carbohydrate was consumed. In the last period, 400 gm. banana, the carbohydrate in the feces came to only 0.9 gm. There was a higher calorific intake than in the control period but also less bread and cereal than in either of the other two. The bananas used in this case and in Case 6 were very ripe. The peel was allowed to assume a dark brown color before the fruit was served and would be considered too ripe by most persons. The flavor of these bananas was excellent.

Case 6.—N. G., aged 3 years, boy weighing 25 pounds. Arthritis of the right ankle. The bowel movements and stools were normal throughout the test. This subject paralleled Case 5 and the same routine was followed. The patient was discharged before the last period could be completed. The appetite of this boy was very good and he consumed his full required quota. The average carbohydrate content of the feces for the control period was 1.2 gm. per day and for the 200 gm. banana days, 2.16 gm. More carbohydrate was consumed in the latter period than in the former.

Case 7.—L. C., aged 3 years, boy weighing 27.6 pounds. Empyema. The stools contained a large number of segments of tape worm (*Saginata*); they were frequent, soft, and bulky. The stool of March 24, was bloody. There were no stools voided April 17 and 22. At the end of the first day an enema was given; no treatment for the omission of defecation on the 22d; two stools voided on the 23d. At no time was there constipation and no stools from this patient were firm or well formed. The regular ward diet was used as the basal ration and the banana was added to this in place of some of the cereal and potato. The boy had an unusual appetite and desired more food than was allowed during the test. At no time was there any discomfort attributable to the diets. The bananas used were very ripe. There were three banana test periods. One day's dietary in the last period was the following: 7 a. m., cream of wheat 50 gm, bananas 100 gm., milk 200 c.c.; 10:30 a. m., bread and butter 1 slice, milk 200 c.c.; noon, cream soup 100 c.c., bread and butter 1 slice, bananas 200 gm., milk 200 c.c.; 3 p. m., bread and butter 2 slices; 5:30 p. m., bread and butter 1 slice, bananas 100 gm., milk 200 c.c. This gives approximately 1,500 calories for the day, of which nearly 26 per cent. is derived from the banana. Period of April 11-15: 200 gm. of bananas were consumed per day and 996 gm. of feces were voided with a carbohydrate content of from 3.2 to 8 per cent., and averaging 5.75 gm. per day. Period April 16-20: 150 gm. of bananas were consumed per day and 534 gm. of feces voided with a carbohydrate content of 1.52 to 3.21 per cent. The stool for the 18th was very soft and that for the 19th liquid. The stool for the 20th, which was almost liquid, reacted acid to litmus, one of the very few acid stools of the whole series of experiments. The average amount of carbohydrate eliminated in these stools was 1.85 gm. per day. This figure is low in comparison with the others of this test and it must be assumed that there was an appreciable loss through fermentation of the fecal matter. There was also a suspicion at the time the feces were collected and analyzed that there had been a loss of stool wholly or in part, but no evidence to this effect could be obtained. Period of April 21-27; control period; no bananas were served. The child was allowed to eat freely of the regular ward diet. The stools were not as soft as those of the preceding periods. The percentage of carbohydrate in the stool ranged within narrower limits, 1.58 to 1.77. The average daily elimination of carbohydrate in the feces was 1.35 gm. Period of April 28 to May 7: 400 gm. bananas were given. The amount of carbohydrate in the stools was irregular, varying from 1.07 to 3.2 per cent. There were five very

soft stools for the first two days; the subsequent stools were more normal in appearance. The lower per cent. of carbohydrate in the stools fell in the middle of the period. The daily average carbohydrate elimination for this period was 2.55 gm. This case is included in the report because it gives a very different impression from all of the others, one less favorable to the banana. The tapeworm is in this case a disturbing factor in the interpretation of the analytical results, as we can have no assurance that its glycogen content is evenly distributed throughout the sample, and the segments of the parasite were far from being uniformly passed in the stools.

CASE 8.—F. T., aged 2 years, boy weighing 28 pounds. Acute bronchitis. The control diet consisted of farina, bread, butter, potatoes, rice, egg, apple, cream, and milk. The carbohydrates ingested each day came to 120 gm. Four days constituted a period. The banana period had baked underripe and raw ripe bananas, besides the same amount of bread and butter as in the previous period, and an extra portion of butter which was worked into the mashed baked banana. The milk in this second period was the same as before; the cream was doubled. This period gave 140 gm. of carbohydrate, 30 derived from baked bananas and 75 gm. from raw bananas. All the food prescribed was taken and the child was well satisfied with his meals. There was no evidence of discomfort or disturbance of any kind. The stools were soft and yellow and defecations regular. The carbohydrate in the stools was remarkably constant, from 0.75 to 0.81 gm. per day. The lower figure fell within the control period. The apparent utilization of the banana was better than 99 per cent.

CASE 9.—M. S., aged 3½ years, girl weighing 25.25 pounds. Osteo-arthritis (arthritis deformans). This experiment was synchronous with and similar to that in Case 8, with practically identical results.

CASE 10.—G. B., aged 4½ years, boy weighing 30.75 pounds. Enlarged bronchial lymph nodes. The control diet consisted of farina, bread, butter, soup, egg, potatoes, cream, and milk. These were given to the subject for the days February 18 to 21 and March 10 to 13. Ripe raw bananas were given with this control diet from February 22 to March 9, and baked underripe bananas replaced the raw bananas from March 14 to 16. There was an interference with the experiment in that calomel and salts were administered from March 8 to 11. During this time the stools were frequently liquid and decidedly reducing to picric acid before acid hydrolysis. The estimated carbohydrates for the stools for these days indicated a carbohydrate elimination of from 4 to 8 gm. per day, consisting almost entirely of reducing sugars. Following this period of purgation the fecal carbohydrate dropped to 1.56 gm. per day for the remainder of the control period and to 1.28 gm. for the banana period. All of these stools were soft and at times almost liquid. The stools prior to the calomel treatment were of firm consistency and in the control period yielded only 0.6 gm. carbohydrate per day, and in the banana period from 0.4 to 1 gm.

CASE 11.—H. S., aged 4½ years, girl weighing 34 pounds. Acute parenchymatous nephritis. The determination of the nitrogenous waste products in the blood of this patient gave high figures within the limits accepted as normal. The patient was later discharged as cured. Constipated; nearly all stools followed either enema or suppositories. The diet prior to the test was restricted as to salt and fluids; otherwise like the regular ward diet. The appetite was subnormal. The following was the menu for the first four days: morning, farina 60 gm., ripe raw bananas 100 gm., cream 1 tablespoonful, and 200 c.c. milk; noon, baked underripe bananas 75 gm., cocoa 200 c.c., steamed rice 60 gm.; evening, ripe raw bananas 100 gm., cream 2 tablespoonfuls, and milk 200 c.c. The second period of four days was the same, with baked potatoes in place of the baked bananas. In the third period the ripe bananas were also omitted, and bread and butter given in their place, the rice being

replaced by cabbage and lettuce. The total daily amount of carbohydrate in the feces for these periods came to 0.5, 1.4, and 1.8 gm., giving no excess attributable to the banana.

TABLE 5.—A SUMMARY OF THE NUMERICAL DATA IN THE DIGESTION EXPERIMENTS ON BANANAS WITH CHILDREN AS SUBJECTS

Case	Period	Carbohydrate Intake, Gm.	Carbohydrate in the Feces, Gm.	Apparent Utilization of the Carbohydrate, per Cent.	Remarks
1	Control	210	3.1	98.5	Yellow bananas used, sliced in last period
	Banana	320	8.6	97.3	
	Control	210	3.2	98.7	
	Banana	320	7.0	96.8	
2	Control	210	4.1	98.1	Same as in Case 1
	Banana	310	5.7	98.3	
	Control	210	2.6	98.8	
3	Control	142	0.7	99.5	Brown spotted bananas
	Banana	147	0.8	99.5	
4	Control	142	0.1	99.0+	
	Banana	147	0.2	99.0+	Same as Case 3
5	Control	90 ?	2.6	97.2 ?	
	Banana	120 ?	2.9	97.7 ?	Golden yellow bananas
	Banana	130	0.9	99.3	Brown spotted bananas
6	Control	992	1.2	98.6	
	Banana	120	2.2	98.2	Brown spotted bananas
7	Control	120	1.4	98.9	
	Banana	...	1.9	....	Fully ripe bananas in the three periods but different amounts
	Banana	...	5.8		
	Banana	154	2.6	98.3	
8	Control	120	0.9	99.2	
	Banana	140	0.7	99.5	Baked bananas
9	Control	120	0.8	99.4	Baked bananas
	Banana	140	0.8	99.4	
10	Control	120	1.6	98.7	
	Irregular	120-131	4.0-8.0	93.5-97.0	Purgatives given
	Banana	131	1.3	99.0	Brown spotted bananas
	Banana	124	1.0	99.2	Baked bananas
11	Control	120	1.8	98.5	Baked potatoes
	Banana	124	0.5	99.6	Baked bananas
	Banana	131	1.4	98.8	Brown spotted bananas

For more convenient comparison of the material in these studies the numerical data have been arranged in tabular form (Table 5). The essential factors are the amount of carbohydrate intake and the amount of carbohydrate output in the stools, shown in the first two columns of numbers. The third column gives the calculated proportion of ingested carbohydrate not accounted for in the feces. The difference between the amount in the stools and that consumed is commonly considered as that which has been assimilated and used by the organism. Since the carbohydrates of the banana are compared with other vegetable foods fed under identical conditions, this apparent assimilation may reasonably be supposed to give a true criterion of the nutritive value of the banana.

Feces contain small amounts of substances which may reduce picric acid after hydrolysis other than those derived from the carbohydrates of the food, but these are so nearly negligible that they may for the present be left out of consideration and the total reduction charged against the unassimilated carbohydrates. In the cases that have been studied the difference between banana and the foods with which it has been compared are very small indeed and in most instances to the advantage of the banana. In the first two cases the bananas used were in the late yellow stage of ripeness; that is, they did not have the full golden tint or any browning on the peel. Such bananas are usually considered ripe and possess the favorite flavor. The fruit at this stage does not give the blue color reaction when treated with iodin, but by careful teasing and with the aid of the microscope starch can easily be demonstrated as present. Microscopic examination of the stools of these two children revealed starch grains. From theoretical considerations one would expect somewhat lower utilization of the carbohydrates than the data indicate, possibly some raw starch being converted into its simpler components in the intestines. In all of the following cases the banana when fed raw was fully ripe; that is, there was no trace of the greenish cast to the yellow of the peel and there was at least a beginning of the brown coloration. In cases 7, 8 and 9 the bananas were decidedly brown and considered over ripe and unfit by the maids of the hospital. The almost complete utilization of these is apparent from an inspection of the table. There is a more complete assimilation in cases of constipation than when the bowels are loose. This is made evident by comparing 3 and 4 with 7, and the days of violent purgation with the normal days in Case 10. Most of the children were constipated. There was no positive evidence brought out that the banana as such was the cause of constipation any more than any other carbohydrate food, and in only two instances (including the one reported in this paper, 7) is it suggested that the banana may have the opposite effect. The impression of the physicians who observed the cases in the ward was that the banana test meals tended to produce a condition of constipation.

Baked bananas have been fed to adults and their apparent utilization compares well with that of the baked potatoes, being over 98.5 per cent. Baked bananas both from the green and the early yellow stage were fed to Patients 8, 9, and 11 of this series and compared with both raw ripe bananas and with the control foods, including baked potatoes, showing somewhat better utilization than the raw bananas and the other foods; but the differences were always so slight as to be practically negligible.

There was no evidence that the feeding of raw ripe bananas in large amounts for periods up to two weeks caused any deleterious

effect on a young child. The periods were too short to show much influence on the weight, though in Case 7 there was a small gain. In only one case was there any disturbance that might be attributed to the banana. This was Case 1, a delicate, shy, Italian girl who was not very fond of the fruit. Though very tractable and friendly to those in charge she would not admit a distaste for the banana until the end of the experiment. In the fourth period the bananas were fed in thick slices instead of mashed, as was otherwise done, to see whether poor mastication would decrease the utilization of the carbohydrates. A slightly lower utilization is indicated by the analytical data. The last noon meal was regurgitated about one-half hour after eating and in the regurgitate were lumps of the banana. No other symptoms devoloped and the child seemed in as good a condition as ever. It should be noted that in this period the bananas were in the stage of late yellow before the development of the full golden tint or before the beginning of the brown spots.

Bananas were also given to a child younger than any of those reported above. This was a mongolian idiot that would not take his meals voluntarily. The child was 19 months old and fed largely on milk and semiliquid food. He was kept on milk and baked bananas for eleven days and on nothing but ripe raw bananas for another ten days. The amount of bananas which the nurse could get into this child ranged from 175 gm. to 550 gm. a day. When milk was prescribed none was consumed on several days and the most taken without loss through regurgitation was 225 c.c. It was easier to get this boy to take the bananas than any of the other foods offered. There were no unfavorable results from the exclusive ripe raw banana diet.

SUMMARY

The banana is a useful fruit that can with profit enter liberally into the child's dietary provided it is fully ripe or well cooked. If eaten baked in the yellow stage of ripeness or if eaten raw when fully ripe, the banana makes a delightful and highly nutritious article of food. Its composition does not warrant the use of the banana as the main component of the child's dietary, but it can compete well with other fruits and is decidedly to be preferred to candies. The nutritional value is relatively high, approximately one calory per gram of pulp; and its carbohydrates, when it is fully ripe or cooked, are not less assimilable than those of cereals and potatoes. In the raw food the digestibility is directly proportional to the ripeness of the fruit. There is no positive evidence that the banana influenced bowel movements. In the many tests there was no suggestion whatever of any deleterious effect from consuming large amounts of fully ripe bananas. Prolonged use of the underripe fruit, on the other hand, developed

undesirable symptoms. Ripeness can be readily determined by the color and texture of the peel as indicated by Table 3. In ripening, the peel changes in color from its original deep green to a dark brown. In this change the color of the banana passes through several yellow stages which are generally taken to be signs of ripeness, but a yellow banana is not necessarily a ripe banana and if consumed raw while it still has a green cast to the yellow color, as is frequently done by children on the streets, the availability of its carbohydrates is comparatively low and the effect on the child's digestive system injurious. The banana ought not to be eaten raw until after the brown spots begin to appear. It is at this stage a full golden yellow and in its most attractive appearance. A completely browned skin is not in itself a sign of overripeness and such fruit should be judged by the texture of the pulp. The brown color of the peel, however, should not be confused with the darkening due to bruises. An injured banana is soon invaded by moulds and yeast cells both through the abrasions and the broken end, so that the banana "finger" should not be broken from the "hand" or stem but cut off in such manner as to leave a good margin between the cut surface and the pulp. The banana properly handled and allowed to ripen to its most beautiful color and texture is a wholesome food, uncontaminated by dirt and pathogenic germs even if purchased from the push cart in our congested streets.

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